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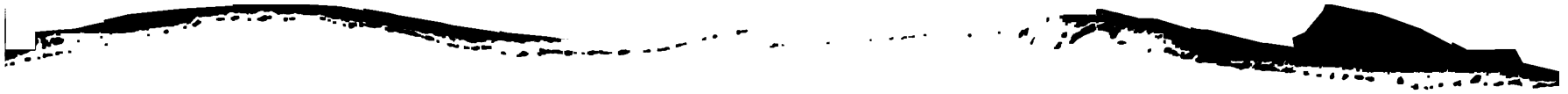

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OF

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EDITED BY

THOMAS L. STEDMAN, M.D.
NEW YORK CITY

IN TWENTY VOLUMES

VOLUME XI

DISEASES OF THE NERVOUS SYSTEM

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DISEASES OF THE CEREBROSPINAL AND SYMPATHETIC NERVES.

BY

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DISEASES OF THE CEREBROSPINAL AND SYMPATHETIC NERVES.

C. A. Reed 1897

In describing the diseases of the nerves of the human body it will not be possible entirely to avoid trespassing upon the domains of other writers. This is for the obvious reasons that, first, the nerve trunks are an integral part of the central nervous system and have either their origins or their ramifications within the great cerebro-spinal axis; and second, that many diseases are not sharply delimited by nature to this one particular territory, but extend their ravages to the nerve centres; or even, originating in these centres, may ultimately involve the nerves and display in them some of their most characteristic symptoms. It is nevertheless true that by a general consent the various diseases of the nerve trunks are now fairly well demarcated from those of the central nervous system, and it is rather the exception than the rule for them to involve the central structures. I shall therefore follow the custom of excluding diseases that are properly located above the nuclei of the peripheral nervous system, but I may occasionally invade these nuclei for purposes of illustration or thoroughness.

It becomes necessary, first of all, in such a paper as this, to devote some space to a general discussion of the anatomy, physiology, symptoms, and pathology of the nerve trunks. In such a brief general discussion the advantages will be seen of not limiting the description exclusively to those trunks, but of pointing out how the subject is interwoven with the wider subject of the whole nervous system.

In the second division of the subject each particular nerve will be taken up, and its diseases will be described and its pathological relations pointed out.

GENERAL ANATOMY OF THE NERVES.

In order to understand the general anatomy and physiology of the nerve trunks and their ramifications, it is absolutely necessary to study them briefly in their relationships with the rest of the nervous system. This has become especially so since the discoveries of Golgi

and his followers have led to an entirely new conception of this system. As Hill [1] has said, Golgi's method of silver staining has thrown more light upon the structure of the nervous system than all other methods, microscopical, physical, or experimental, put together. It is essential, therefore, to avail ourselves of these results in order to gain a proper conception of the position of the nerve trunks in the nervous economy.

The essential element of the nervous system is the nerve cell. This nerve cell is an absolute anatomical unit. By this is meant that it exists by itself, independent of all other nerve cells or of other tissues. It has no *continuity* of structure with any of these, but only *contiguity* of structure. Golgi's and other allied methods prove this fact. They demonstrate that the nervous system is only an aggregation of distinct and separate nerve cells, having mutual relations but not anatomical continuity. This is in opposition to the view, which so long prevailed, that the nervous system was a sort of continuous texture, in which the processes of one cell anastomosed with those of other cells. It can now be clearly demonstrated that there is no such anastomosis. It thus becomes all important to study the nerve cell as the seat of all neurological phenomena.

Taking as a type one of the large pyramidal cells of the cerebral cortex, we distinguish at a glance two integral parts—the *body* and the *processes* of the cell. These together form the anatomical unit now called the *neuron*. Schaefer proposed restricting the term neuron to the axis-cylinder process, but this proposition has not been accepted. Waldeyer suggested the term neuron for the whole nerve cell, including its axis cylinder, which latter some have proposed to call the *neuraxon*. Waldeyer's plan is that which is now quite uniformly adopted.

The body of the neuron is always the seat of the nucleus, in which again is seen the nucleolus. This nucleus is both absolutely and relatively larger in nerve cells than in other cells (Schaefer). Its function is apparently largely nutritive—a fact of first importance to the neuropathologist. If, for instance, any portion whatever of the neuron be cut off from that portion which contains the nucleus, the part so cut off will inevitably degenerate and ultimately die. The protoplasmic contents of the neuron present a reticular appearance, especially well marked in cells stained by Nissl's method. In its nutritive function and its dependence on the nucleus, the neuron has distinct *amœboid* characteristics, *i.e.*, it resembles that lowest unicellular animalcule called the amœba.

The processes of the neuron, as can readily be seen, are of two kinds. The more important of these is the *axis-cylinder* process.

In the embryo this is the first process that appears, antedating, in the human embryo, the other kind by several weeks. His (quoted by Déjerine[2]) observed that the axis cylinder in the human embryo appeared in the first month, but that the dendrons did not appear until the tenth week. This does not seem to confirm the theory that these latter subserve nutrition entirely. The axis cylinder is distinguished by its smooth surface and its perfect regularity. Déjerine also points out that it can be told from the other kind of processes by a cone of origin. It is apparently a simple prolongation of the cell and its contents; if there is any differentiation of structure in it from the cell, it has not been demonstrated. Its function is limited entirely to transmitting impulses. In the case of all neurons the axis-cylinder process gives off branches in its course, which are called *collaterals*. These are identical in structure with the main limb. They and the main limb end in tufts of fibrils, which are called *arborizations*. The axis cylinder and its collaterals are prolonged into the nerve trunks, and thus constitute the nerve fibres.

The second process, or collection of processes (for there are usually more than one), are different in several important respects from the first kind. They are larger, coarser, and more numerous than the axis cylinder. They were called by Deiters the protoplasmic processes, but they are now usually called, after His, *dendrites*, or, after Schaefer, *dendrons*. They do not form an axis cylinder. Their surface is not smooth, as is that of the axis cylinder, but is marked in some instances with small bead-like swellings (especially in the embryo), or with small, regularly distributed prominences, as though the surface were frosted. The dendrons usually send off branches, in some instances very many (as in the cells of Purkinje), and this branching begins not far from the body of the cell. The function of the dendrons is claimed by some biologists to be purely nutritive, but this is not probable; they must transmit impulses, especially in the numerous instances in which they are interlaced with the terminal arborizations of the axis cylinders of other cells. The purely nutritive function of the dendrons is rendered impossible also by the fact that some important cells have none of these processes—the unipolar cells of the spinal ganglia, for example. In all cases the dendrons terminate by free ends, never by anastomosis.

There are many varieties of neurons, according to their location and function in the nervous system. Schaefer proposes to distinguish between *dendric* and *adendric* cells, according to the presence or absence of dendrons; and again, to distinguish cells according as they have one or more axis cylinders. Again, some neurons have much longer axis cylinders than others; and finally, in some neurons the

axis cylinder arises not from the body of the cell, but from a dendron. Considering these facts, Déjerine observes three kinds of cells: (1) Cajal's cell, in which the axis cylinder may be multiple, but always arises from a dendron; (2) Golgi's cell, in which the axis cylinder is single, but very short and divided into numerous ramifications; (3) Deiters' cell, in which the axis cylinder is also single, but long and divided into collaterals. The cells of the second type were claimed by Golgi to be always sensory, those of the third type always motor; but this is probably erroneous.

The terminal arborizations of the axis cylinders and of their collaterals are interlaced in some instances with the dendrons of other neurons, but in other instances form a kind of network around the bodies of these neurons. In whatever way they are disposed their ends are always free.

As already said, it has been held by some, especially by Golgi, that the function of the dendrons is to take up nutritive fluid for the support of the cell body, and that the axis cylinder alone has a purely nervous function. In support of this view, Golgi claimed to have demonstrated that the dendrons were often found in connection with the capillary system. This view has been opposed by others, prominent among whom are Cajal and Lenhossek, who maintain that the whole neuron is nervous in its function, and that if there be any differentiation it consists in the possible fact that the dendrons are *receptive* and the axis cylinders are *cellulifugal, i.e.*, conduct impulses outwards (Déjerine). This latter view would seem to be in accord with the observed facts of cytology, for as a rule the function of nutrition in cells is not differentiated in the manner claimed by Golgi.

We must next, for our purposes in this paper, view briefly the plan in which these various kinds of neurons are grouped in order to form the nervous system.

The nervous system, central and peripheral—to use an expression of Déjerine—is constituted merely by a series of neurons more or less prolonged. The accompanying diagram from that author explains this fact more fully than can words. Before analyzing it in detail, it is important once more to emphasize the fact that each individual neuron is an independent anatomical *unit*. In consequence of this fundamental fact, as is seen in the diagram, there is no anastomosis, and consequently there is no network of nerve fibres in the sense in which Gerlach used the term and which was so long adopted, *i.e.*, a network of living anastomosing fibres, constituting a sort of widespread textile organon, receiving and originating the peripheral nerve fibres. So absolutely is this simple anatomical unit, the neuron, regarded by some as the unique basis of all neurological phe-

nomena, that authors like Déjerine even deny that there is any distinction functionally between a motor and a sensory neuron, but claim that the difference in action results merely from the difference in anatomical connection, the one being distributed to muscle and the other to skin. While it is difficult to conceive how all differentiation between neurons can be denied, especially when we consider the higher psychical functions, still it is a fact that, so far as their pathological reactions are concerned, they are all apparently alike. This fact concerns us here especially, because we are about to describe the diseases of the peripheral nervous system only. But it will be necessary first to understand the anatomical relation of this system to the cerebrospinal axis.

It will be seen, by reference to the diagram (Fig. 1),* that the motor side of the whole nervous system consists of but two neurons—a central and a peripheral neuron. The central neuron (N_1) has its cell body in the cerebral cortex (*EC*). It is a large pyramidal cell, distributing a few dendrons in its immediate neighborhood and sending a very long axis cylinder downwards. This axis cylinder is, in fact, one of the fibres of the crossed pyramidal tract, and ends in the spinal cord by arborizations, which interlace with the dendrons of the second or peripheral neuron (N_2). This peripheral neuron is, in fact, one of the large multipolar cells in the anterior horn of the spinal cord; it, as we have just said, has dendrons interlacing with the arborizations of the axis cylinder of the central neuron. It also sends out a long axis cylinder, which has but few if any collaterals, and distributes its end-arborizations in a muscle. This axis cylinder forms one of the component fibres of the nerve trunk.

On the sensory side we see that the efferent tract is composed of *three* neurons. The first, or peripheral neuron (N_1), is of special interest, because it originates entirely outside of the central nervous system. Its cell body is located in the spinal *ganglion* of the posterior root. It is usually represented, as in the diagram, as a unipolar cell without dendrons. Its single axis cylinder divides into a T-shaped double branch, one limb of which is distributed in the skin (*Ep*); the other passes by way of the posterior root into the spinal cord, and there, in the posterior column, divides, one branch passing downwards, the other upwards. Both these branches send out collaterals, which have various terminations. Thus one collateral is shown in the diagram to distribute its arborizations amidst the dendrons of the peripheral motor neuron (the multipolar cell of the anterior horn); another goes to a neuron in Clarke's column (*CC*),

*I wish to acknowledge especially my obligations to Déjerine's excellent work in preparing this brief description.

which sends its axis cylinder up by way of the direct cerebellar tract to the cerebellum (N_2 with dotted line). The ascending branch terminates around or near the second or intermediary sensory neuron ($NG + B$). This second or intermediary neuron has its cell body in one or other nuclei of the columns of Goll and Burdach in the

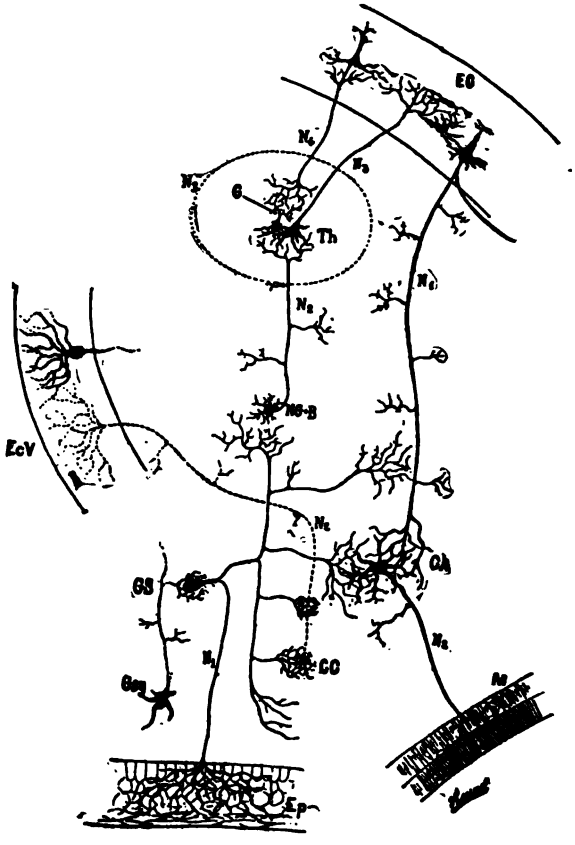

FIG. 1.—Diagram Showing Relations of the Nerves to the Nerve Centres. (Déjerine.)

medulla oblongata. It sends its axis cylinder up to the thalamus (*Th*). Here its arborizations interlace with the third or central sensory neuron (N_3), which sends its axis cylinder to the cerebral cortex, to end by arborizations which interlace with the dendrons of small cortical cells, which in turn interlace with the pyramidal or central motor neuron. Thus, as it were, the circuit is completed. The diagram represents a few other collateral connections—as, for instance,

a large collateral arising from the pyramidal cell just as the axis cylinder leaves the cortex. This is probably sent through the corpus callosum to the other hemisphere. Other connections of the sensory neurons through the thalamus (*Th*) with the cortex are also seen. But with these we are not here specially concerned. The figure is, of course, absolutely and merely diagrammatic, and serves its purpose to illustrate the relations of the nerve trunks to the central nervous system.

The peripheral sensory neuron is of special interest, because of its mode of origin and the light which it throws upon the development and anatomy of the nerves of special sense. As seen in the embryo chick, duck, etc., the neuron, from which develops the sensory nerve running from the skin through the posterior root into the cord, grows in the ganglion of the posterior root. It is originally bipolar, sending out an axis cylinder from each end or pole. In the course of development this bipolar neuron gradually changes into a unipolar one. The cell gradually assumes a position to one side of the two axis cylinders, which thus appear as one continuous fibre, the cell being attached to it by a process of its own. This is explained differently by Jakob, who claims, from analogy with the neurons of special senses, that the so-called axis cylinder going to the skin is not embryologically an axis cylinder at all, but a *dendron*. Lenhossek demonstrated that the cells of these peripheral sensory neurons in earthworms are located in the epithelium of the skin, whence they send each an axis cylinder direct to the central nervous system. As we ascend the animal scale we find these peripheral sensory neurons retreating, as it were, farther and farther inwards, until when we reach the vertebrates the cell bodies are grouped in the ganglia of the posterior roots, far within the parietes of the body. These facts in comparative anatomy are shown in the accompanying diagram (Fig. 2). Their significance with reference to the nerves of special sense will be shown when we come to describe the diseases of those nerves.

From the foregoing description it is now easy to understand that the nerve trunks are merely collections of axis cylinders arising from the cells of neurons, whether these cells are in the nerve centres or in the ganglia outside of the nerve centres. These axis cylinders, however, are enclosed in protective sheaths, and the nerve trunks are supplied also with connective tissue and blood-vessels, with all of which it is necessary to be familiar.

We owe largely to the investigations of Ranvier our exact knowledge of the sheaths of the axis cylinders. These are two in number. The first of them to appear embryologically is the outermost one, or

sheath of Schwann. This is formed by cells which, very early in embryonal life, apply themselves closely to the axis cylinder. They become immensely elongated and attenuated (His), and by the end of the first month they form a sheath which completely encloses the axis cylinder. This sheath, however, as proved by Ranvier, is not continuous, but is broken up into segments, each segment representing one of the original enclosing connective-tissue cells. These segments are limited by constrictions, the annular constrictions or nodes

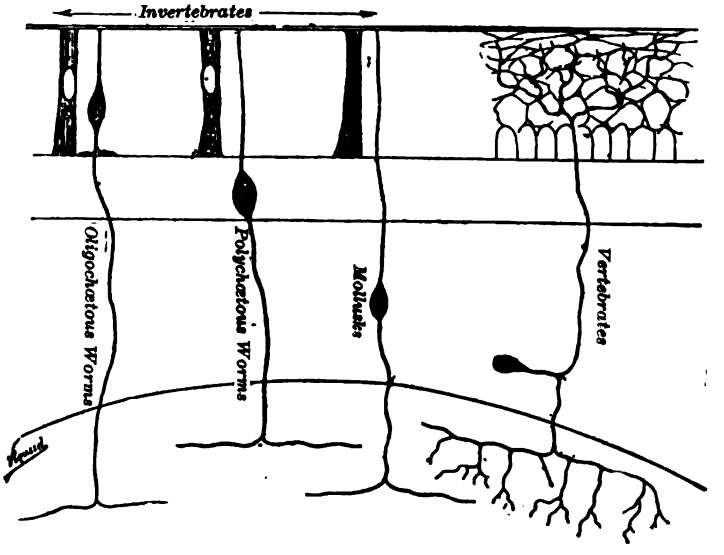

FIG. 2.—Varying Position of the Cells of the Peripheral Sensory Neurons. (Déjerine.)

of Ranvier (*a*, Fig. 3). Each segment contains a nucleus at about equal distance from the annular constrictions.

The second sheath is the *myelin* or medullary sheath (*m*). This consists of a perfectly homogeneous substance interposed between the sheath of Schwann and the axis cylinder. In life it is transparent, but it rapidly undergoes post-mortem changes. It is this myelin that gives the nerves their white appearance. The reactions of this substance to various reagents, as osmic acid, nitrate of silver, etc., have been described and depicted in great detail by Ranvier, Lautermann, Golgi, and others. Many of these appearances, as Kölliker has said, are probably artefacts caused by the reagents. The anatomy of the nerve fibre is shown in Fig. 3.

The structure of the *axis cylinder* itself has been the subject of some dispute. According to Déjerine it is a mass of fibrils; accord-

ing to Leydig, Nansen, and others, it is merely a continuation of the reticulum composing the cell body of the neuron; and according to Schaefer, it is a bundle of distinct tubes, which spread out at the periphery in their distribution, each preserving its identity. According to the view of all of these, it is at least probable that the axis cylinder contains a protoplasmic fluid, either within these tubular fibrils, or between them, or in the meshes of the reticulum, according as either view is adopted. Lenhossek[3a] in his recent work depicts a nerve cell in which the axis cylinder is not drawn as though composed of fibrils or of reticulated tissue, but is of homogeneous substance, in marked contrast with the body of the cell.

Non-Medullated Fibres.—Remak demonstrated the presence of non-medullated fibres. They are found especially in the sympathetic system in vertebrates, although not exclusively in that system. Thus the fibres of the olfactory nerve are non-medullated. These fibres, or fibres of Remak, are also found in other parts of the cerebrospinal axis. Déjerine says that they have a fibrillar structure, such as he believes is the case in the medullated fibres, and like these they end in arborizations, but never by anastomosis.

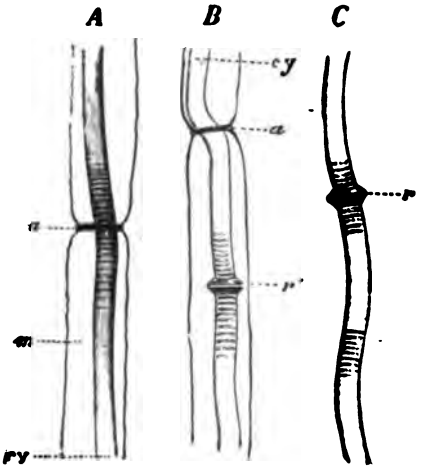

FIG. 3.—Anatomy of the Nerve Fibre. (Déjerine.)

The Connective Tissue of Nerves.—As the seat of many of the pathological changes that underlie the diseases of the peripheral nervous system, it is highly essential that the connective tissue should be thoroughly understood. This tissue has two functions especially to perform; one is to bind together and to protect the nerve fibres, the other to support the blood-vessels. Both of these functions are the causes and the occasions for some of the most striking and profound of the morbid manifestations that occur in the nerve trunks.

A nerve trunk, as already explained, is a collection of axis cylinders, each of these being encased in its protective sheaths. These axis cylinders, however, do not run all together in one long tube, but are grouped in small divisions, each of which is called a fasciculus. Thus a nerve trunk consists of a collection of fasciculi, each fasciculus holding a number of nerve fibres. The connective tissue which forms

these subdivisions and holds them together is, therefore, divisible into several kinds, which have been variously named.

The connective tissue which forms the *wall* of each fasciculus is a firm lamellated tissue (*gl*, Fig. 4). It forms a solid tube, as it were, excepting that here and there it is perforated by minute blood-vessels, which carry nourishment to the nerve fibres within. In smaller nerves, in which each fasciculus contains but a few nerve fibres, the lamellated sheath is quite thin and delicate; but in the larger nerves, in which the fibres in each fasciculus are numerous, this sheath becomes much denser and thicker by the increase of its concentric lamellæ. Thus in the sciatic nerve the walls of the fasciculi are quite dense.

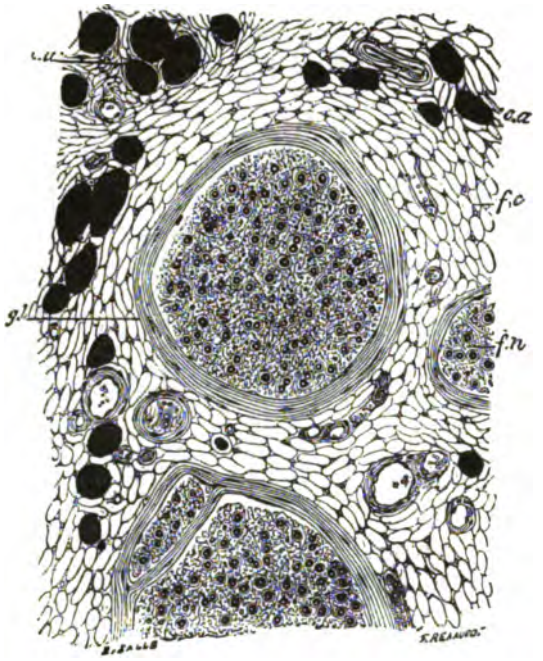

FIG. 4.—Cross Section of a Nerve; *f.n.*, nerve fasciculus; *gl.*, sheath; *f.c.*, connective tissue; *c.a.*, adipose cells. (After Ranvier.)

The connective tissue *within* each fasciculus serves the purpose of a supporting membrane for the nerve fibres. It is composed partly, according to Ranvier, of laminæ which insinuate themselves between the nerve fibres, and subdivide the fasciculus into still smaller but not very distinct compartments. These laminæ are really extensions inwards of the lamellated tissue of the wall of the fasciculus. They also conduct and support minute blood-vessels. The intrafascicular connective tissue is also composed in part of fine fibres, and does not differ materially from the connective tissue of other parts, except that it is without elastic fibres (Déjerine). It serves entirely as a supporting membrane.

Finally, there is the connective tissue *between* or among the fasciculi. This is ordinary connective tissue, which, according to Ranvier, has its fibres running almost exclusively in a longitudinal direction,

instead of crossing in all directions. It supports the main blood-vessels of the nerve trunk, and has embedded in it a certain amount of adipose tissue.

The Blood-Vessels of the Nerves.

The nerve trunks are copiously supplied with blood-vessels. These arise from many branches, but usually from only one, or at most a few main trunks. In most instances the artery passes obliquely to the nerve trunk, and then continues for some distance on the outside of the sheath, giving rise to many branches. After penetrating the sheath it divides into two branches, one of which passes upwards, the other downwards. The interfascicular branches are numerous (see Fig. 5). Small branches penetrate the fasciculi, within which they give origin to a network of capillaries. Ranvier also described a capillary system in the perifascicular connective tissue.

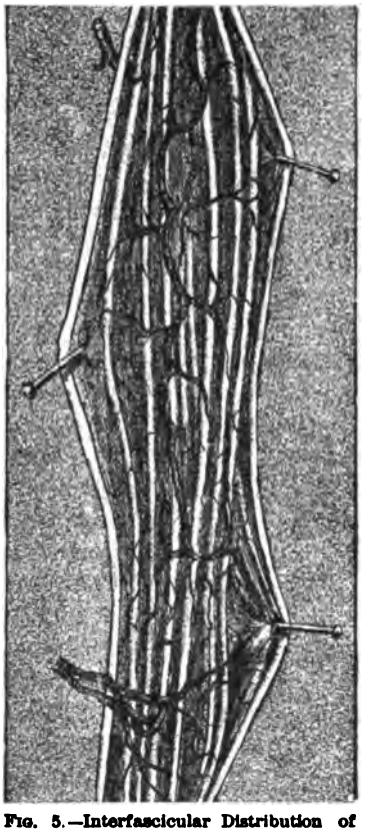

Fig. 5.—Interfascicular Distribution of Arteries in a Nerve Trunk. (Quénu and Lejars.)

The veins follow in the main the course of the arteries.

The lymphatic vessels as such do not originate in the fasciculi, but in the perifascicular connective tissue. The lamellated walls of the fasciculi form a system of lacunæ, which act as lymph channels and communicate with the lymphatic vessels in the perifascicular connective tissue (Ranvier).

Termination of the Nerves.

The fundamental fact to be remembered is that the nerves terminate by free ends at the periphery, just as it has been shown already that the neurons terminate in the central nervous system. The view, formerly held so commonly, that there are terminal sense organs in the shape of cells or other structures, continuous with the structure of the nerves, is now known to be erroneous. The so-called corpuscles of Langerhans, for instance, have been proved by the silver methods to have no identity with the nervous tissue. In the *skin* the

sensory fibres (peripheral sensory neurons) terminate by free arborizations. According to Déjerine, these fibres, still covered by their medullary sheaths, ramify under the basal membrane of the papillæ, and there give off fibres which are non-medullated. These latter, presenting slight varicosities, ramify freely, and terminate *between* the cells of the Malpighian bodies.

In the *mucous membrane* the arrangement is practically the same as that in the skin. The small non-medullated terminal fibres present the same minute varicosities, giving the nerve twig a beaded appearance.

This general plan of termination is preserved in all nerves of common sensation. Modifications, which occur in various structures, do not radically change the type. All of them are marked by this bead-like appearance of the non-medullated fibres, which is produced by small varicosities on the nerve twigs. In all cases the endings are free.

In the *glands* the terminal fibres form networks around the glandular culs-de-sac, from which networks fine end-fibres are detached which attach themselves directly to the secreting cells without the interposition of any membrane. It must not be understood, however, that these networks are formed of anastomosing fibres—they are rather merely interlacing fibres.

On the *motor* side the termination of the nerves is also by free arborizations, but these are confined in the voluntary muscle to a small territory or bulb, known as the *motor plates*, described by Rouget, by Krause, and by Ranvier. These motor plates are small swellings, each one on a distinct muscular fibre. They present nuclei and are covered by a layer of sarcolemma. The nerve fibre, still medullated, penetrates this sarcolemmatous sheath, its own medullated sheath being lost within the motor plate, in which its fibres end by a free arborization. It is thus seen that the type of terminal arborizations is preserved here perfectly, the only distinction being in the organ in which this arborization is effected.

GENERAL PHYSIOLOGY OF THE NERVES.

The physiology of the nerve cell, or rather of the neuron, is of cardinal importance now, since it forms a basis for the whole science of neurology. Unfortunately it cannot be said that this physiology is further advanced than its infancy.

The physiology of the neuron is somewhat vaguely described by physiologists as the elaboration of a "nerve impulse." The exact nature of this nerve impulse is not thoroughly apprehended; and, in

fact, the term, it is to be feared, is too generalized to convey very definite ideas or accurate knowledge. We have already seen that Déjerine holds that this impulse is identical in all neurons, that the only differentiation consists in the character and function of the structure to which the terminal arborizations are distributed. This is reducing all neural phenomena to the older *neurility* of Vulpian; but when the complexity of psychical functions is considered, it is quite inconceivable. It therefore is essential to recognize that neurons are differentiated in function, and that this function cannot be all comprehended under one term. According to Donaldson,* the prevailing idea seems to be that this nerve impulse is generated as the result of certain chemical changes in the cytoplasm of the cell. It seems to be accompanied with certain electrical phenomena, but it would be too much to say that it is identical with them. It can be excited by mechanical, thermal, electrical, or chemical irritants. It is not essential that the irritant should be applied to any particular part of the neuron; at whatever part it is applied the nerve impulse spreads out from it in all directions; or, if the impact is made to a branch, as the axis cylinder, the impulse extends in opposite directions. This impulse seems to be limited to the neuron itself; that is to say, this impulse, when *artificially* excited by an external irritant, does not transcend the limits of the neuron. Thus, when the fibres in the anterior root of the spinal cord are stimulated with electricity, there is not found by the electrometer any evidence of nerve impulse at the cut end of the cord somewhat cephalad of the irritated root (Donaldson). This is explained by the fact that the nerve fibres in the anterior or ventral root are really axis cylinders from the large multipolar cells in the anterior horns (peripheral motor neurons), and that consequently the impulse does not transcend the limits of those neurons, which limits are in their dendritic branches, which are distributed in their immediate neighborhood in the cord. The function of these motor neurons is, of course, to send motor impulses towards the periphery, and this they do also when artificially excited; but it is not their function to send impulses upwards through their dendrons into the arborizations of the central neurons, and accordingly they do not send these artificially excited impulses upwards either. But as they are constantly receiving normal or physiological impulses from above through their dendrons, it seems to follow that the path of transmission is normally from the arborizations of the contiguous upper cells into the dendrons of the lower-lying cells, and that this path is not reversed for impulses excited by electrical irritation. This is in accord with the opinion of some biologists, that the dendrons are *receptive* organs and that the axis cylinder is a *transmitting* organ. It

seems to be proved, however, that nerve impulses can travel through a neuron in a direction *reverse* to the physiological one; as, for instance, in the peripheral sensory neuron. If, for instance, the cut end of the cord be stimulated in the posterior column, the impulse is observed in the posterior root on the *distal* side of the ganglion. Here, however, the impulse is still confined to the one neuron, which has its cell in the ganglion. Donaldson supposes that while the body of the cell is excitable, yet the dendrons are especially so—in other words, that they are adapted especially for the function of receiving impressions. It must be remembered also, as Donaldson observes, that the conditions of observation are such that it is not yet practicable to isolate a nerve cell completely for experimental purposes, and hence the question has been raised whether experimentally the neuron after all is not dependent upon irritation of the arborization of contiguous cells for its stimulation. *A priori*, there does not seem adequate ground for such an assumption, if we accord to neurons an inherent quality of excitability. In other words, we may suppose that each independent neuron is artificially excitable, although physiologically it may, as a rule, depend upon the stimulus which it receives from the cell lying next in series to it. This question is, after all, only a physiological quibble. If, for instance, it is claimed that the cell body is not excitable except through the arborizations of another cell, the question of excitability is only transferred from one cell to the other, because whether the cell of one neuron is excitable or only the arborizations of another, in either case it results that one of these neurons is excitable. The peripheral sensory neurons certainly are excitable without intermediation of another neuron. For the pathologist there seems to be no difficulty in supposing that the neurons are excitable by morbid agents anywhere in their course. This certainly seems to be the teaching of pathology.

Schaefer and Horsley have attempted to show that neurons have a rhythmical discharge, and that the rate of this is about ten times a second. It is demonstrated by experiment that the nerve impulse travels at the rate of about one hundred to one hundred and twenty feet per second; in the visceral nerves it is only about twenty-five to thirty feet per second.

It seems to be still an obscure question how nerve impulses pass physiologically from one neuron to another. Whether this is by actual contact of the arborizations of one neuron with either the dendrons or the cell body of another, or whether in the possible absence of such contact the nerve impulse in some unaccountable way leaps across this minute space, or is conducted by the intervening neurogliar tissue—these are still vexed questions. That there is actual movement

of the neuronal endings, and that these consequently are applied to and withdrawn from the contiguous neurons, has indeed been suggested; but there is nothing in actual observation to support such a theory. The inference that a neuron acts like an amœba and may put forth pseudopods, is entirely fanciful. In embryonal life, indeed, it has been claimed by biologists that the young cells just developing from the neuroblasts are migratory, and the prolonged growth of their processes, especially their axis cylinders, implies, of course, some progression through space; but to infer from this that a power of lively movement remains in the neurons during adult life is not warranted. It can be equally said of other embryonal cells that in adjusting themselves in the formative period of life they have some power of movement, or rather of being moved, but this does not imply that they retain an active amœboid faculty during mature life. All that we can say at present is that there seems to be an appreciable delay in the transmission of an impulse from one neuron to another.

A distinction, important to the neuropathologist, must be made between the irritability and the conductivity of a neuron or its axis cylinder. A fibre which for any reason may have lost its power to respond to external stimulants, may nevertheless retain its power to conduct an impulse from the cell body to the terminal arborizations. This is seen especially in nerve fibres that are undergoing regeneration after injury.

Other data concerning the functions of the neuron have been and are being gathered by physiologists, but many of them relate to phenomena which are still obscure and are not essential to our study in this place.

The *nutrition* of the neuron is dependent in some way, just as in the amœba, upon the nucleus. Any portion of the neuron, either of its body or of any one of its processes, will infallibly die if cut off from that portion of the cell body containing the nucleus. The exact vitochemical changes that take place in this act of nutrition are unknown. This is still the prime secret of nature. The method of absorbing nourishment is apparently through the cell wall by a process of osmosis. In this process of nutrition the material is stored which liberates the force which, as we have seen, is vaguely called the "nerve impulse." In fact, this *is* the nutritive process probably; for it scarcely seems conceivable that in such a simple body nutrition should be a separate process from the storage of material to make nerve force. Hence the idea of some (Golgi *et al.*) that the dendrons are alone concerned with absorbing nutritive material and that the axis cylinder is alone a true conducting nerve organ, is not generally

accepted. The only differentiation seems to be that the dendrons receive impulses and the axis cylinder transmits them. In favor of the view that nutrition and energizing are practically one and the same process (or different phases of it), is the fact that a nerve cell which does not receive its due allowance of stimulant, and hence does not give forth impulses, is very soon seen to atrophy. Thus if the peripheral sensory neuron is deprived of its cutaneous branch by an amputation, it atrophies. It seems, therefore, that each repeated act of expending force must be followed by renewed nutritive activity, and thus the vigor of the cell is maintained. According to Donaldson, this is true to such an extent that in a series of neurons, if one fails to transmit impulses to the cell next in the series, this latter cell sooner or later wastes.

From the facts already stated arises one of the basal laws of neuropathology—the Wallerian law. According to this a nerve fibre (axis cylinder) when cut off from its nutritive centre (cell body with nucleus) degenerates. This law was announced before its true meaning was recognized; but it is now thoroughly understood by referring it to the trophic functions of the cell. The nerve fibre in the nerve trunk is part of that organic whole called a neuron. When it is cut off, either by mechanical injury or by disease, from the cell body and its nucleus, it degenerates and ultimately perishes. This fact is seen so commonly in numerous diseases of the cerebrospinal nerves that it is highly essential that the physiological law upon which it depends should be understood.

In addition to this peripheral degeneration, there is observed under some circumstances a *central* atrophy of the neuron when its axis cylinder or its other connections have been interfered with. To be seen in its entirety, however, this atrophy must be seen in young animals. It is observed especially well when the injury has been inflicted during intra-uterine life. This atrophy may proceed even to complete disappearance of the whole neuron, cell body and all. Von Gudden made use of this fact to establish a new method of investigation (Edinger). He observed that, if in very young animals the axis cylinder were broken off and the cell body dislodged from its place (*i.e.*, its dendritic connections disturbed [?]), the remaining portion of the neuron—that is to say, its cell body with the nucleus—atrophied and entirely disappeared. By artificially removing the end organs of neurons, as, for instance, by extirpating the eye, he was enabled to follow up resulting atrophies in the brain. These changes probably depend upon a law, already mentioned, that the nutrition of a neuron keeps pace with its energizing, and therefore that if anything interferes with the more or less constant stimulation of the cell body by

repeated impressions from without, this cell body will tend to deteriorate and waste. This, of course, would apply to the sensory cells more than to the motor in case of an amputation of a limb. But even in the case of a motor neuron, if its axis cylinder is cut off and the cell's connection with its muscle is destroyed, its function must be almost abolished. Donaldson suggests that the struggle for existence may be a factor in the causation of this atrophy, since it seems that this struggle for nutriment is quite marked among embryonal cells. Edinger examined the spinal cord from a man who had had an intrauterine amputation of the left forearm, and found marked atrophy in the left anterior horn of the cervical enlargement. It is important to recollect that this atrophy is much more marked when the injury has occurred in embryonal or early life than when it has happened in the adult. It is more marked also the nearer to the trunk the amputation is.

In addition to these pathological changes in nutrition, Hodge demonstrated that there is a recognizable change within strict physiological limits. In other words, he showed that in a condition of fatigue the neuron presents distinct visible changes, which evidently represent a consumption of plasma in the elaboration of force. These changes were noted especially in the cells of the spinal ganglia of sparrows, in cerebral and cerebellar cells in other birds, and in those of the antennary lobes of bees. The method, in brief, was to observe these cells after they had been subjected to a full day's work, and then to compare them with similar cells from other similar animals after a night's rest. Similar effects can also be produced by prolonged stimulation of any of these cells by the faradic current. This has been done by Hodge[4] in the cat and in the frog. The changes noted are, briefly, shrinking and in some instances vacuolation of the cytoplasm (the protoplasm of the cell), with lessened power to stain; and shrinking and crenation of the nucleus, with loss of its open reticular appearance. The nucleolus also is smaller than normal. Slight changes also are noted in the nuclei of the sheaths. Vas and Mann[5] found a preliminary swelling of the cell after a brief period of stimulation. Mann says that during rest several chromatic materials are stored up in the nerve cell, and that these are used up by it during its activity.

The significance of these trophic changes in the neuron as a result of sustained activity is considerable to the neuropathologist. They are probably similar to what occurs in some states of prolonged nerve exhaustion. It is probable, as the axis cylinder is but a prolongation of the cell body, that its nutrition also suffers somewhat from long-continued exertion, but this is not demonstrable as yet un-

der the microscope. Hodge says that the method of Kupffner and of Bovere, by osmic acid and acid fuchsin, demonstrates the essentially fibrillar construction of the axis cylinder, but he seems to have made no observation of changes in these fibrillæ as a result of his experiments. It has been found that prolonged stimulation (for five hours) by electricity does not abolish its conducting power (Bowditch, quoted by Donaldson), but this does not prove that its nutrition is not impaired in a corresponding degree to that of the cell body. In diseases of the peripheral nervous system we possibly meet with instances of nerve tire and nerve exhaustion, in which the underlying condition may be identical with those minute anatomical changes originally described by Hodge. In such instances it would, of course, be difficult or even impossible to regard the condition in the nerve trunk (axis cylinder) as distinct and apart from the condition in the cell body, since these form one anatomical unit, the neuron. Hence in treating of the diseases of the nerve trunks, it may be necessary to note this fact occasionally, and perhaps sometimes to transcend the strict limits of the peripheral nervous system.

From the general facts already stated with reference to degeneration and the Wallerian law has come much special knowledge of the course of individual nerve fibres. This is so especially of the roots of the spinal nerves. These roots are two in number. They are usually called in human anatomy the anterior and the posterior root, but they are better called the ventral and the dorsal root—the distinction observed by morphologists in general. The degenerations resulting from section of these roots are as follows: When the posterior or dorsal root is cut between the ganglion and the union with the anterior or ventral root, the cut end next to the ganglion remains normal, but the cut end away from the ganglion degenerates; moreover, in the nerve trunk formed by the union of these two roots is found a large number of degenerated fibres, which may be traced as sensory fibres to their arborizations in the skin. When the posterior or dorsal root is cut between the ganglion and the spinal cord, the cut end next to the ganglion (just as in the preceding case) remains normal, but the cut end away from the ganglion degenerates and the degenerated fibres extend for some distance in the cord (posterior columns). It is thus seen that the degeneration in the dorsal or sensory root runs in one instance centrifugally and in the other centripetally, and not, as was formerly claimed, always in the direction of its normal impulses—which is centripetal. But in either case mentioned the degeneration is always in the fibres which have been cut *away* from the ganglion, the obvious reason for which is that the ganglion contains the cell body of the neuron. When, on the other hand, the an-

terior or ventral root is cut between the cord and the union of the two roots, the cut end next to the cord remains normal, but the cut end away from the cord degenerates; and, moreover, in the nerve which is formed by these two roots a large number of degenerated fibres is found, and these can be proved to be motor fibres by the fact that they are distributed to muscular fibres. In both the ventral and dorsal roots, however, there are a few fibres that are an exception to the rule. Thus if the dorsal root be cut between the ganglion and the cord, a very few fibres are found *not* degenerated in the cut end next to the cord. These are evidently *efferent* fibres running out by the dorsal root from neurons whose cell bodies are probably located in the anterior horns. Likewise, if the anterior or ventral root is cut, while most of the fibres in the cut end away from the cord degenerate, a very few fibres are found *not* degenerated in this end; and these are probably fibres which come from neurons in the ganglion on the dorsal root, and which, instead of proceeding down the nerve trunk, turn at the union of the two roots and pass upwards to the ventral or anterior root, thus endowing this root with its so-called "recurrent sensibility." For it is found that the *distal* cut end of the ventral root—which is undoubtedly motor—has nevertheless a marked sensibility when irritated, and this, of course, it can derive only from the posterior or dorsal root (Foster).

With reference to their functions the roots of the spinal nerves have been called respectively *sensory* (for the posterior or dorsal root) and *motor* (for the anterior or ventral root). There are objections to these terms, however, especially in the fact that all nerve fibres running *out* of the central nervous system are not necessarily motor, and all fibres running *into* it are not sensory. For instance, some of the former run into glandular tissues, and some of the latter are for merely reflex arcs, and do not necessarily influence the sensorium. Hence more satisfactory physiological terms are found in *afferent* for the dorsal, and *efferent* for the ventral roots. These terms indicate that in the former case the nerve fibres convey impulses *into* the central nervous system, and in the latter that they convey impulses *out* of this system. In the first case the impulses are centripetal, in the second they are centrifugal.

Electrotonus.

The axis cylinder of a neuron, or the collection of axis cylinders forming a nerve trunk, gives a series of definite responses to electricity; and this quality of responding to an electrical stimulus is called the *electrotonus* of the nerve. This electrotonus is manifested by a variety or a series of responses, according to the form or mode of elec-

tricity which is used, and these responses can be formulated under a set of laws.

The responses are not the same for muscular tissue as for nervous tissue—a distinction that must be carefully observed. In the human body, of course, it is not possible artificially to separate the muscle from the nerve for purposes of experiment and study, and as the irritability of the nerve is the direct cause of the response in the muscle, the two are apt to be associated in mind as giving identical responses. But the electrotonus of muscle, when entirely separated from the nerve, is quite different from that of the nerve, and this fact is demonstrated for us in the human body not infrequently by *disease*. Hence the electrotonus of the nerves is an important object of study in many diseases of the peripheral nervous system.

To *faradism* the response is as follows: With one electrode on the nerve trunk an active response occurs every time the current is *broken*. To prove that this response occurs only when the current is broken, and not when it is made, it is only necessary to regulate the automatic interruptor of the ordinary faradic battery, so that it acts very slowly, or, better, to move it by the finger at stated intervals; and it can then be readily seen that the muscle does not react when the contact is made, but only when it is broken. This is according to a law which will be explained later. The response to faradism is quick and strong, and this is its *modal* characteristic. If the interruptor is allowed to vibrate rapidly, the muscle is thrown into a state of almost tonic rigidity; it is "tetanized," as the expression is. In other words, it does not become completely relaxed between each faradic stimulation, and this is because the faradic shocks are in such rapid sequence that the muscle has no time thus to relax. In this reaction to faradism the muscle reacts only because stimulated through the nerve; in other words, muscular tissue by itself is not excitable by the faradic current.

To *galvanism* the reactions of a nerve trunk follow a definite law. It is customary to study these reactions as they occur in motor nerves, because these nerves excite visible responses in the muscles to which they are distributed; hence these responses are easily studied. But it is important to bear in mind that the law of electrotonus is the same for both sensory and motor nerves, although not so readily appreciated in the former as in the latter. When a motor nerve is stimulated by electricity and a response is excited in the muscle, the nerve is not acting as a mere conducting wire to the muscle; in other words, the muscle in these circumstances is not responding to an electrical impulse which has been conveyed to it by the nerve. On the contrary, it is not necessary that any of the elec-

tricity should reach the muscle; the nerve responds by virtue of the special power of the neurons to generate a nerve impulse under excitement, and it is this impulse which excites the muscle. Hence the farther from the muscle the nerve is stimulated, *i.e.*, the nearer to its cell bodies in the cord, the more active the response in the muscle, which would not be the case if the response depended on transmitted electricity along the nerve to the muscle (Bennett). Another proof that in these circumstances it is not the muscle but the nerve which is excited by the electricity, is the fact that when muscular tissue is entirely separated from its nerves and directly stimulated with electricity, its responses are different in some important particulars from those which it exhibits when its nerves are excited. Still another proof is the fact that a muscle is especially excitable at the point where the nerve enters it—its so-called motor point.

The character of the response to galvanism is sudden, tense, and quick, just as it is to faradism. But these responses are due to alterations in the current strength; without such alterations no response occurs to galvanism. Thus after a current has started to run through a motor nerve there is no continuous contracture of the muscle all the while it is running; the muscle contracts only when an alteration in current strength occurs. If this alteration is sudden and complete, as when the current is made and broken, the contraction of the muscles is prompt and conspicuous. The quick, prompt character of the response to electricity is called, as above stated, the *modal* characteristic. The responses to the alterations in current strength, *i.e.*, to the making and breaking of the current, are called the *serial* or qualitative responses, and these have also a definite character.

These serial responses follow the law of Pflüger. According to this law an exposed nerve, as in a frog, responds to the *negative* pole when the current is *closed*, and to the *positive* pole when the current is *opened*. The negative pole increases excitability, the positive pole diminishes it in the neuron.

In order to render this important physiological law still more clear, it can be studied diagrammatically. Some confusion would be avoided if the terms *ascending* and *descending* currents were abolished, and if the action of the current were studied with reference to only one electrode at a time. The diagram (Fig. 6) represents two nerves, A and B, each with a muscle attached. In A the current from a battery is applied with the anode near the end of the nerve and the cathode near the muscle. If an irritating substance, as salt, be applied at R while the circuit is open, the muscle responds actively; but these responses become weaker or entirely disappear as soon as the current is turned on. The explanation for this is that the posi-

tive pole with its sedative influence puts the nerve in a state of lowered excitability at and near the irritated point, and consequently the nerve cannot respond to an irritant; but as soon as the circuit is *opened* once more, *i.e.*, as soon as the sedative effect is removed, the muscle responds promptly. If, however, the irritation be made at R′, the response of the muscle is increased at the *closure* of the circuit, because at this end of the nerve the negative or stimulating pole acts, and is not interfered with by the anode. In B the current is applied in an opposite direction. If the irritant, salt, be applied at *r* and a weak current be turned on, the response to the irritant is increased, because the irritant acts in conjunction with the cathode or stimulating pole, while the anode is too weak to oppose an effectual barrier. But with a strong current the result is different; the muscle does not respond, and this is because the anode has now increased in power sufficiently to deaden the sensibility of the nerve and prevent the passage of the impulse to the muscle. Thus Pflüger's law is simplified by the statement that it depends upon the sedative action of the anode and the stimulating action of the cathode upon the neuron, according as these are placed with reference, in the motor nerves, to the nerve impulse and the muscle.

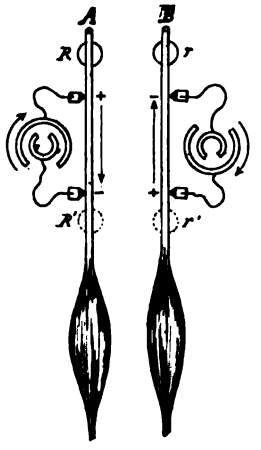

FIG. 6.—Method of Testing the Excitability in Electrotonus. (After Landois and Stirling.)

In order to test these responses in the human body it is necessary, of course, to apply the electrode to the nerve through the skin. The results are not exactly the same as when the naked nerve is experimentally tested in the lower animals. Such exceptions as occur are probably caused by diffusion of the current. But one electrode should be applied over the nerve; this is called the *active* electrode; the other, or *indifferent* electrode, may be placed on the sternum or elsewhere on the surface at a point where it cannot influence the nerve to be tested. The positive pole is called the *anode*, the negative pole the *cathode*. Under these conditions it is possible, of course, to stimulate the nerve in *four* different ways, *i.e.*, by closing the current or by opening the current, with either the cathode or the anode on the nerve. It is found on the human nerve that the cathode, at closure of the current, is the most stimulating; consequently with a very weak current this may be the only response, and it will certainly be the first in the series. It is called the cathodal closure contraction, or

CCC. The next in the series to excite a response is the anode, but at the opening instead of the closing of the current; hence this is called the anodal opening contraction, or AOC. The third in the series to excite a response, with a gradual increase of current strength, is the anode at the closing of the circuit; hence anodal closure contraction, or ACC. The last, and usually only with a very strong and painful current, is the cathode at the opening of the circuit; hence cathodal opening contraction, or COC. These may be called the classical *serial* or *qualitative* reactions of the neurons to electricity. Most healthy nerves respond to a definite strength of current,

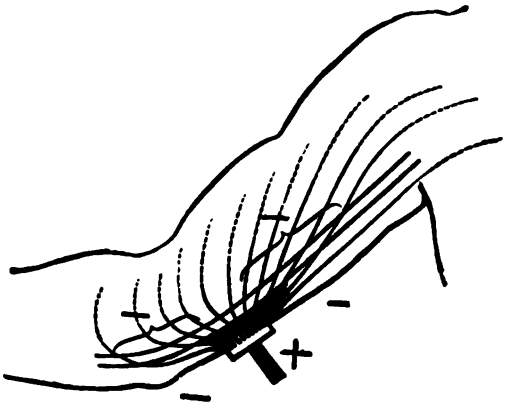

FIG. 7.—Diagram Showing Diffusion of Current Leaving a Nerve.

although nerves in the human body differ among themselves as to the required strength, probably because of physical characteristics, depth below the surface, etc. Within normal limits the nerve is said to have a *quantitative* reaction; but this may be increased or diminished by disease. Why these variations occur—why, in other words, the neuron responds with different degrees of vigor to these variations in the application of the current—is a question which it is not possible to answer. It acts so because of some inherent quality in the cytoplasm, or some essential but obscure quality in the electric current, with which we are not familiar.

In the human subject, however, with the current applied to the nerve through the skin, the results are not always the same as those just given; sometimes the anodal closure contraction—ACC—equals if not even exceeds in force the anodal opening contraction—AOC. The only reason we can give for this exception is that the diffusion of the current through the nerve must in some way modify the results. Thus according to De Watteville what seems to be an ACC is actually

a CCC, the cathode acting at the closure of the circuit, but in the zone of greater diffusion, and hence not exciting as strong a reaction as when acting in a more concentrated form. This is illustrated in the accompanying diagram (Fig. 7). Here the anode is applied over the ulnar nerve. The anodal influence or zone is marked +. But because of the diffusion of the current it escapes from the nerve in a much wider zone, and this of course is cathodal, and is marked −. Now when a contraction occurs at the closing of the circuit at this pole, it is probably not an *anodal* but a *cathodal* effect, and thus the law of Pflüger is not invalidated, as it would seem to be at first sight by the apparent reaction of the anode at the *closure* of the circuit. If we suppose the current to be reversed, we see at once why it is so difficult to obtain the COC. In this case the anode would be acting in the zone of greater diffusion, and as the opening contraction really depends upon it, this could be obtained only with a current of great strength. In other respects the series is as in the exposed nerve; the cathodal closure is always the most exciting, and the cathodal opening is the least so. A response to the latter, in fact, is so difficult to obtain, and the procedure is so painful, that it can practically be put out of count.

The formula for the normal reactions is usually stated thus:

$$CCC > ACC > AOC > COC.$$

The sign > indicates that that reaction is greater towards which the sign expands—thus the formula, CCC > AOC, indicates that the cathodal closure contraction is greater than the anodal opening contraction. If the two symbols are joined by the sign of equality (=), this indicates that these two reactions are equal in force. Thus the formula AOC = ACC, indicates that the anodal opening contraction and the anodal closure contraction are equal, as is the case usually in the normal human nerve. Thus the formula for the normal human nerve, as tested through the skin, is as follows:

$$CCC > ACC = AOC > COC.$$

It has been stated that the reaction occurs only at the moment when the current is either closed or opened, and not while the current is running through the nerve. This is so, however, only within certain limits; to very strong currents, running continuously, there may be a continuous tetanic contraction, and this is called the *duration tetany*. Its symbol is DT (De Watteville).

The reactions thus far stated are those of the neurons; the muscular responses are secondary and entirely subordinate to the excitation of the nerves. But the *muscular tissue* has its own way of responding to electricity. This can be tested when this tissue is apparently en-

tirely cut off from nervous influence, as happens sometimes by the action of certain drugs (curare), and, more important still, by the action of certain diseases. These reactions, in brief, are as follows: To faradism, the muscular fibre will not react at all. It seems that this tissue is not sufficiently excitable to respond to the exceedingly brief shock of the faradic contact. When a muscle seemingly reacts to faradism, it does so in fact because it is still attached to living and functionating neurons, and is receiving impulses from them and not directly from the current. Hence the apparent muscular response to faradism is always an important indication that the nervous tissue is still healthy. To galvanism the muscular tissue, when freed entirely from nervous influence and still healthy itself (?), responds at first to even a milder current than does the neuron; in other words, it is rather more excitable. This is called a *quantitative* increase. It is especially notable if the shocks are *slowly* repeated, *i.e.*, if the *quantity* of electricity is augmented. Instead, however, of this response being sudden and quick, it is distinctly slower and more sluggish than it is when the muscle is excited through the nerve. This is called the *modal* phenomenon. It may even amount to a tetany (duration tetany). The serial or *qualitative* reactions to galvanism are the same in the muscle as they are in the neuron, as long as the muscle is attached to the healthy nerve, but after it is separated these reactions change and constitute the "reactions of degeneration" (p. 74).

It must be held in mind, of course, that nerve and muscle are so closely related that it is difficult to prove that the muscular tissue is really *healthy* when cut off from its normal nervous connection. Thus it may be a question whether the quantitative increase to galvanism is not really due to irritation of the intermuscular nerve endings, caused by degenerative changes in them. This will be discussed again in relation to the reactions of degeneration, when the qualitative changes also will be described.

Besides the variations and exceptions already noted in the law of electrotonus of nerve and muscle, the following is of importance: To obtain the AOC it is sometimes necessary to permit the current to run for a certain appreciable time before breaking it. It seems necessary to get the nerve thoroughly under the current's influence before it will respond to the stimulus at the breaking of the contact. Again, after repeating this procedure a few times, the AOC gradually disappears; the same is true of the COC. But this is not observed of either the CCC or the ACC. These facts are explained by referring again to Pflüger's law of electrotonus of nerve. According to this law the cathode excites the neuron, but the anode dulls it. Hence the cathode causes a reaction at the closure of the current,

while the anode can cause a reaction only at the opening of the current, because this opening of the current acts as a removal of a sedative influence, which permits the neuron to regain its normal point of excitability, and this is marked by a reaction. Consequently by prolonging this sedative influence for a time the rebound of the neuron, as it were, is more marked, but repetitions of this sedative action seem to exhaust the neuron for the time.

Modes of Sensation.

A sensory neuron conducts at least three distinct kinds or modes of sensation. These are, first, the *tactile* sense; second, the *temperature* sense; and third, the *pain* sense. Some authorities would add a fourth, or *muscular*, sense; this, however, may not be a true sense, but merely an affection of the motor neuron itself, appreciable by consciousness.

In addition to these *general* or common modes of sensation, there are also the *special* senses. For these latter senses certain neurons have been set apart or differentiated to subserve a special purpose. They are only sensitive each to a particular kind of impression; for instance, to the impression made respectively by light, sound, and odorous and sapid substances. It is customary to divide sensation into *common* and *special;* but it is not easy to say exactly in what the distinction consists. Common sensation, says Dana, is that which is referred to the body itself, while special sensation is always referred to an object outside the body; thus pain is a common sense, but touch, like sight and hearing, is a special sense. But is this distinction radical? The painful prick of a pin is certainly referred to its object. The truth is, the distinction is unimportant. The functions of these special senses will be described more in detail when the diseases of their nerves are described. To the biologist they present important problems with reference to the evolution of their neurons from the ordinary neurons of common sensation. For the present it is sufficient to say that they are evidently thoroughly differentiated, and do not subserve any of the functions of ordinary or common sensation.

It is an interesting question why and how the same neuron apparently conducts the three modes—touch, pain, and temperature. This problem is made more obscure by the undoubted fact that some diseases (syringomyelia and some forms of neuritis) dissociate these modes of sensation, abolishing one or two and not the other. We are not exactly concerned here with the purely psychological question of what constitutes pain.* We are considering now the several modes of sensation, one of which is that state called physical

pain. As there is no satisfactory evidence that there are separate peripheral neurons, one for each distinct mode of sensation, we are forced to the conclusion that each peripheral sensory neuron is endowed with the faculty of conducting all these separate impulses. There does not seem, from the physiological standpoint, to be any difficulty in conceiving the neuron to be thus endowed; the cytoplasm in the motor neuron is well known to react to more than one kind of irritant (electricity, acids, salt), therefore in the sensory neuron also there may be the power of reacting to different kinds of stimulants. Such are, at any rate, the plain physiological facts.

It is proper to state, on the other hand, that some authorities claim that the peripheral sensory neurons *are* differentiated for these several functions. Thus Landois and Sterling' state that there are *two* functionally different fibres—one for pain, the other for touch—but they give no proof for such a claim. Jakob says that it is not determined whether different tracts for the various qualities are present, but he assumes that it is probable. Goldscheider (quoted by Landois and Sterling) goes even so far as to claim that there are *four* special kinds of cutaneous nerves—one for heat, one for cold, one for pressure, and, lastly, one for touch. It is notable that those who claim the existence of such special nerves give no histological evidence of them, and that they vary as much among themselves as do Landois and Sterling on the one hand, and Goldscheider on the other. Ferrier has given in part a more rational explanation; he says that the facts are more readily explainable on the theory that there are differences in the peripheral organs and in the receptivity of the nerves to various forms of external agencies. He thinks that disease may so affect the common sensory paths as to render them unable to convey one kind of impression while still able to convey another. As to a difference in the end organs, here assumed by Ferrier, it is sufficient to recall that these sensory end organs are the arborizations of the neurons, and that there is no evidence of any differentiation in them, at least in external form. As to his second theory, that nerves of common sense may also be affected by various stimulants, as heat, pain, etc., this seems to be the nearest in accord with histological facts. It is quite possible to conceive, as already said, that the peripheral sensory neuron is able to respond to various stimulants and to conduct a variety of impulses (as touch, temperature, and pain), and that in some pathological conditions one or other of these powers may be abolished. As Ferrier says, we cannot even profitably speculate as yet upon the possible changes in the molecular condition of the axis cylinder upon which such differences may depend.

The *tactile* sense is not distributed equally to all parts of the skin. It is more acute in some regions, as the tip of the tongue, the tips of the fingers, the palms, the face, etc. It is least marked in the back. It is entirely absent from the viscera, according to the few accurate observations that have been made by means of fistulæ, etc., but these observations require to be confirmed so far as the mucous membranes are concerned. The tactile sense is excited only through the end organ, according to Landois and Sterling; but this statement is not in strict accord with observations made on amputated limbs. In such cases there is usually a phantom limb, in which subjective sensations of touch, as well as pain and muscular sense, exist and are referred to the periphery. Still it is true that ordinary irritation of a nerve trunk does not excite tactile sensations referable to the periphery.

Too strong stimulants of any kind applied to the periphery no longer cause simple tactile sensations, but excite pain; in this instance, the one mode of sensation apparently passes into the other. When pain is once established, the tactile sensation is no longer appreciated as such. This seems to be a proof that these two modes of sensation are only variations in the reaction of one and the same kind of neurons.

Tactile impressions may be both enumerated and located within certain limits by the mind. Both acts, enumeration and localization, are probably purely mental acts, and therefore not modifications of the sensory act proper.

By the act of localization the mind tells what part is touched, *i.e.*, what group of neurons is affected. This, as already said, is apparently a pure act of perception, hence an act of the brain, and not properly an act of the peripheral sensory neurons themselves. It may be disturbed, however, by disease of the peripheral nervous system; this is evidently because, in such an instance, the disease interferes with the conduction of impressions to the brain or conducts altered or abnormal impressions; hence the mind, receiving either no impressions or only abnormal ones, is not able to conclude properly as to location. It fails simply because it has not its proper stimulus. This is proved still further by that singular paradoxical action of the mind in which it mistakes entirely the part touched, and may even locate it on the opposite side. This, however, is not usually due to peripheral disease. According to Landois and Sterling, the sense of locality (or perception, more properly) is more acute the greater the number of tactile nerves in the part; the greater the mobility of the part, as in the fingers and toes; and in the transverse than in the long axis of a limb. When two points are used, they may be distinguished at shorter distances apart if they are applied one after the other, in-

stead of simultaneously; if they are either considerably warmer or colder than the skin; if the points are gradually approximated instead of gradually separated; and if one point is cold and the other hot (although this latter test does not distinguish distance so well). Exercise and a moist skin improve this power of localization, evidently by making the arborizations of the neurons more accessible through the skin; and, for an opposite reason, ischæmia of a part, as that induced by holding the hand aloft, decreases this power.

Weber measured accurately in millimetres the smallest space at which the two separate points of an æsthesiometer can be distinguished in different regions of the body. His table, as given by Jakob, is as follows:

	Millimetres.		Millimetres.
Tip of tongue	1.	Buttocks	39.
Finger tips	2.	Upper arm	65.
Lip	4.	Forearm	39.
Tip of nose	6.5	Hand { volar side	11.
Cheek	11.	Hand { dorsal side	28.
Forehead	30.	Thigh	39.
Chest	44.	Leg or toes	11.
Middle of back	65.		

This table, it is more than probable, is not absolutely fixed. The power of individuals no doubt differs, both in the same individual at different times, and among individuals. Weber's table is accepted and extensively quoted, and no one seems to stop to question its accuracy; but according to my own observations it is far from accurate, especially in the great distance it gives for some parts of the body. Most persons by whom I have tested it have been able to distinguish points much nearer together than indicated in the table. It seems true, however, that the *order* of sensibility of parts, beginning with the tongue, as published by Weber, is in the main correct.

The *temperature* sense, like the tactile sense, varies for different regions of the body. Weber found it less acute on the median line than on either side. The regions of the surface vary in their sensibility to temperature in the following order: tip of the tongue, eyelids, cheeks, lips, neck, and body. This sense is more acute in a large than in a small area. Extreme cold (ice) is more readily mistaken for heat than *vice versa*, but this perhaps only in pathological states. Differences in temperature of very slight degree are more readily appreciated in objects of median temperature (27°–35° C.). Above and below these points temperature gradually grows uncomfortable and merges into pain. This transfer of temperature sense into pain sense is similar to what occurs in the case of tactile sense,

as already noted, and seems to be a further proof that pain sense is only a modification or intensification of one or other of these modes of sensation. According to Goldscheider, two objects, as cylinders, are recognized at much smaller distances apart when they are either hot or cold than when at the ordinary temperature. It is noteworthy that the temperature sense and pain sense are not infrequently abolished together, while the tactile sense remains, as in syringomyelia. If these two senses are but modifications of common tactile sensibility, it seems probable that disease may affect or enfeeble the neuron so as to abolish its power of reacting to heat and pain, without abolishing its power to conduct tactile impulses.

The *pain* sense is always displayed by the sensory neuron when the stimulating action of an irritant applied to it passes certain limits. Hence any and every form of irritant can become the cause of pain—common tactile sense and temperature sense both passing into pain sense by insensible gradation. What the exact limits are between the other modes of sense and the sense of pain is an obscure question, and the molecular changes in the cytoplasm underlying this transition are totally unknown. It is significant, however, that pain usurps, as it were, the place of the others. This seems to be a proof that pain is only a mode of reaction of the sensory neuron to any form of extreme irritation, and that this mode of reaction is a common function of all sensory neurons, and not special to a few.

Pain is referred to the periphery when the nerve trunk is irritated in any part of its course. In this respect it differs from tactile sense. This difference is curiously shown in some cases of irritative lesions which abolish sensation in the distribution of the affected nerve; the sense of pain, excited by the lesion, is referred to the anæsthetic area, so that the patient feels pain in the region in which, to use a paradox, he feels nothing. This is *anæsthesia dolorosa.*

The mind has not always the power accurately to localize pain, especially when this occurs in the interior of the body. As this power to locate pain, just as the power to locate tactile impacts, is a psychic act, the difficulty of locating pain in the interior of the body is probably due to the fact that in health the brain receives very few sensations from these regions; hence it is not trained to observe and locate them. The difficulty of locating pain on and just beneath the surface, and in the limbs generally, is not so marked. When this difficulty occurs in these regions it seems to be in cases of irritation of the nerve trunks rather than of the nerve endings; and may be due to the fact that, first, the lesion is rather subacute; and second, that it may involve more than one trunk, and therefore causes irradiation of pain. Irradiation of pain may also be due in part to a confusion of the

psychic act—the inability of the mind accurately to locate the point of greatest irritation.

When we come to consider certain pathological questions, it will be found that the supreme difficulty in the way of a clear understanding of the paths of sensory conduction in the periphery and cord lies in the fact that disease occasionally dissociates the modes of sensation, abolishing one or some and sparing the other or others. While on the face of it this fact appears to point to the conclusion that these various modes—touch, temperature, and pain—are conducted by separate neurons, and while this conclusion appears to be accepted by many neurologists and neuropathologists, still this theory cannot be said to rest upon secure grounds. Histology does not support it, because, so far as form and terminal arborizations are concerned, there is no differentiation in the sensory neurons. On the other hand, it is quite conceivable that the sensory neuron is excitable by more than one form or mode of stimulant; and that the "dissociation symptom" may be caused by changes that simply abolish the function of the neuron in part instead of in whole.

As we have seen, some parts are more sensitive than others. This is probably because their nerve supply is richer and their arborizations are more numerous than in less sensitive parts. The same may be said with respect to pain. Certain observers claim that some nerves are more sensitive to pain than others, as the fifth nerve, for instance. While this cannot be denied or positively asserted, it is at least possible that the especial sensitiveness, as in the eye, may be due to some peculiarity in the distribution of the ends of the neurons.

The *muscular sense* is the notice which the mind receives of the state of the muscles—their tonicity, contracture, position, and the degree of resistance offered to them. Its exact nature is still the subject of some speculation. It is usually supposed to depend on the sensory neurons. That it is in any way dependent on the motor neurons themselves is not usually even considered, but the possibility of this dependence is not to be ignored. In hemiplegia it is certain that the muscular sense may be affected in some degree, although sensation is not involved.

There are a number of modifications of common sensation, such as hunger, thirst, fatigue, repose, well-being, malaise, and sexual erethism. None of these is dignified as a special sense, and yet they are all specialized to some extent. Certainly they cannot be confused, the one with the other, nor any one of them with any special sense,

The Reflexes.

An important function of the cerebrospinal nerves (the peripheral sensory and motor neurons) is the conduction of the reflexes. It is customary to speak of these as spinal, but they are properly the function of the peripheral neurons, and have only a portion of their course in the spinal cord.

If a sensory nerve ending be irritated, an impulse is conveyed along its axis cylinder, through the spinal ganglion which holds the cell body, into the cord. Here it may take especially one of two courses: one of these leads upwards to the brain, affecting consciousness; the other, by another branch, passes across the cord, and, by way of its arborizations, stimulates the body of a peripheral motor neuron in the anterior horn. An impulse then passes along this motor neuron and excites a contraction in the muscle to which it is attached. This constitutes a simple reflex act, and has properly but two divisions, not three, as usually described; these are an afferent impulse and an efferent impulse. There is no so-called "reflex arc" in the spinal cord, constituting a third division, interposed between the efferent and afferent paths. Our more precise knowledge of the neuron has eliminated such an hypothetical centre. The sensory or afferent impulse may radiate more widely in some instances than in others, and hence excite a reaction in a larger number of motor neurons; thus the extent of the reflex action may vary at times, according to circumstances. This is all that can be meant by speaking of a "centre" or "arc" for a reflex action.

The reflexes are not under the control of the will, except it may be in very slight degree. Most of them, however, are within the sphere of consciousness. The exceptions to this rule (in the iris and ciliary body) will be noted in their proper places.

While not under the control of the will to any great extent, the reflexes may be influenced in a variety of ways. This is so especially of the deep or tendon reflexes. They are subject, for instance, to "reinforcement" (Fig. 8). Jendrassik showed that if a person made a strong voluntary movement just at the moment when the reflex was elicited, this latter was increased in force. This voluntary movement may be made by tightly clasping the hands together, or by clinching the teeth. Weir Mitchell and M. J. Lewis showed that this reinforcement lasts for an appreciable time after the voluntary movement has ceased; and again, that continued reinforcement gradually exhausts or enfeebles the reflex. These observers have also shown that sensory stimulation also increases or reinforces the deep reflexes; thus

if, at the moment of tapping the tendon, a piece of ice or a heated object be applied to the skin, or if the skin be pinched or a hair pulled on any part of the body, the reflex is increased. Faradism applied to the skin, and galvanism passed through the head act in the same way to increase the reflexes. Lombard * demonstrated by experiment that the emotions may increase the force of the tendon reflexes. Such emotions are fear, apprehension, shame, etc. This phenomenon is seen especially well in some hysterical cases.

The reflexes are numerous and distributed widely through the body. The more prominent and easily recognized have received names, but many nameless reflexes doubtless exist; and it is best that they should continue nameless. It is possible, in fact, that every sensory neuron contributes to a reflex act, although some of these may be so slight as to be recognized with difficulty. Two kinds of reflexes are noted especially—the superficial and the deep.

The *superficial* reflexes are such as are excited by slightly irritating the endings of the sensory neurons. The impulse is carried, as already explained, to the body of the motor neuron in the anterior horn and thence reflected along its axis cylinder to the muscle. The irritant may be the prick of a pin, or even in some cases such a slight stimulant as brushing the skin with a feather or with the tip of the finger or of a pencil. The muscle that responds is usually one in the immediate neighborhood, as just beneath the skin of the point irritated.

The following are the commonest and best-known superficial reflexes:

The reflex of the iris to light. This will be explained and its clinical significance pointed out under the heading of diseases of the orbital nerves. It is, of course, not an ordinary skin reflex, but a highly specialized one, and is excited by but one kind of stimulant, light.

The reflex of the dilator muscle of the iris to irritation of the skin of the neck or cheek. The path of this reflex is probably through the sympathetic system, which presides over the dilatation of the pupil. According to Landois and Sterling, dilatation of the pupil is also caused by severe pain, as of torture or parturition, a loud call in the ear, and stimulation of the nerves of the sexual organs. It may be doubted, however, whether all of these are examples of true reflex action. These authorities state that the presence of dilator fibres of the iris is even denied by some observers. This particular question of reflex dilatation of the pupil is still an obscure and vexed one. The observed facts, however, are as given.

The conjunctival reflex. This consists in a violent and involun-

tary closure of the eyelids upon irritation of the exceedingly sensitive nerve endings in the conjunctiva by any foreign body. It persists even in some states of unconsciousness. Its afferent path is along the branch of the fifth nerve that supplies the conjunctivæ. Its efferent or motor path is along the branch of the seventh nerve, that presides over the closure of the lid (the orbicularis palpebrarum). It may consequently be abolished or impaired in certain diseases of these nerves, as in Bell's paralysis of the seventh nerve and in nuclear paralysis of the fifth.

The epigastric reflex is caused by irritation of the skin over the lower part of the side of the thorax, and is shown by a contraction of the upper ends of the recti muscles especially, and of contiguous muscular fibres in the epigastrium.

Very similar reflexes on the trunk are obtained by irritating the skin along the edges of the erector spinæ muscle and over the scapulæ, the muscles contracting being those beneath the skin in these neighborhoods respectively.

The cremasteric reflex is an especially characteristic one. It consists in an active retraction of the testicle, due to the action of the cremaster muscle; and is caused by irritation, as brisk rubbing with the finger nail, of the skin on the inner and upper aspect of the thigh.

The plantar reflex is excited by an irritant applied to the sole of the foot. It consists in a movement of withdrawal of the foot, by bending the knee and hip, and has much the appearance of a voluntary movement. It is best seen when exaggerated in some diseases, especially in apoplectic states; at such times its involuntary character can easily be determined.

The *deep* reflexes are those that are excited by tapping the tendon of a muscle. In order to obtain this reflex it is usually necessary to put the tendon slightly on the stretch. When the tendon is then tapped smartly with the finger ends or any small object acting as a hammer, the muscle contracts promptly and sometimes with considerable force.

The deep reflexes have been the subject of much speculation and some controversy. It has usually been assumed that they are true reflexes, *i.e.*, that an impulse is excited by the tap on the tendon, and that this impulse travels up to the spinal cord, whence it is reflected to the muscle, which responds by a vigorous contraction. If this is so, the deep reflexes are explainable just as the superficial reflexes are, *i.e.*, by the mutual action of a peripheral sensory and a peripheral motor neuron. The chief grounds for believing that the deep or tendon reflexes are genuine are, first, that section of the motor nerve in the rabbit abolishes the patellar reflex (Schultz), and so

does section of the cord opposite the fifth and sixth lumbar vertebræ (Tschirjew, quoted by Landois and Sterling'); and second, that diseases which abolish the function of either the sensory or motor neuron abolish the deep reflexes. Such diseases especially are locomotor ataxia and multiple neuritis. On the other hand, the reaction time for some of the deep reflexes (as the knee jerk) appears to some physiologists to be too limited to allow an impulse to pass first to and then from the cord. Waller found that this time in the case of the knee jerk was but 0.03 second; Eulenburg measured it at 0.032. Those who contend against the tendon reflexes being spinal in origin, claim that they depend upon the natural tonus of the muscle; that this tonus is slightly excited or increased by the tendon being put upon the stretch, and that when this excitement is still more increased by the tap upon the tendon, the muscle responds by virtue of its own inherent tonus. According to this view, the phenomenon is only secondarily dependent upon the nervous system, inasmuch as the tonus of the muscle is maintained by the nerve influence. This fact, therefore, accounts for the influence of nervous diseases upon the tendon reflexes; when they lower the nutrition of the muscle, then the deep reflexes are weakened or abolished; but when they increase this tonus, then these reflexes are correspondingly increased. Whichever view of the origin of the reflexes be correct (and the one that holds the tendon reflexes to be of nervous origin appears the better founded), the clinical significance of these phenomena is the same; hence they must be thoroughly understood.

Although, as already said, the will has but little control over the deep reflexes, yet the cerebral centres in some way exercise apparently an unconscious inhibitory effect upon them. This is shown from a variety of facts. If, for instance, the cerebrum be cut off from the spinal cord by any lesion, which does not act too suddenly at first, the deep reflexes are increased. This is seen constantly in such diseases of the central nervous system as gradually break up the motor paths which run from the cerebral cortex downwards to the various levels of the cord. Such diseases are hemorrhage into the internal capsule, myelitis, especially when transverse and of limited extent, and syringomyelia. Whether this increase is to be considered as due to the release of the spinal apparatus (sensory and motor neurons) from an inhibitory influence of the brain, or whether it is due to increased excitability of these neurons due to some irritative influence exerted upon them by the lesion from above, are debatable points. If there is a true inhibition, it is not probable that this flows from any "inhibitory centre" in the brain—as, for instance, the "centre" described as Setschenow's, which required such an exten-

sive mutilation for its demonstration that its existence may well be considered as yet problematical; but that it is rather due, as Brunton explains, to a wave-like action of nerve impulses, constantly flowing and common to all neurons. It seems improbable that the increase can be due exclusively to irritation caused by the lesion above. The central motor neuron, for instance, must undergo complete degeneration in its distal end in long-standing lesions, and be incapable of conducting any irritative impulses below the lesion.

Inhibition of the reflexes, both superficial and deep, is partially and only for a limited time under the control of the will. The influence of the will upon the reflexes, in fact, is entirely by inhibition, as it has no power to increase them. The will, for instance, can momentarily inhibit the conjunctival reflex, but if the irritation be continued the reflex occurs in spite of the will. Yet this action of the will in this case is not truly so much an act of inhibition as it is an opposing of a countermuscular action—for instance, the action of the levator and other fibres that serve to open the eyelids. We may gather a hint from this that perhaps all inhibition is merely an equalizing, as it were, of the balance of power in the peripheral motor neurons (motor cells in the anterior horns of the spinal cord), and that when this influence is withdrawn the motor neurons in some way act erratically and some in excess of others.

Inhibition, again, may be caused by exciting powerfully a sensory nerve. As already stated, stimulation of the sensory nerves, by ice, etc., increases the deep reflexes, according to Weir Mitchell and Lewis; but, according to other observers, very strong stimulation (pain) may diminish the reflexes, especially the superficial ones (Goltz and others).

Finally, inhibition of the spinal reflexes may be caused by a total transverse lesion of the motor path in the brain or cord, occurring *suddenly*. In other words, a lesion which cuts off the brain influence with the symptoms of shock acts almost invariably as an inhibitory influence. This is a common clinical observation, although from the pure physiological standpoint it is difficult to explain it. Such lesions as hemorrhage in the internal capsule, and hemorrhages into the cord, or crushing of the cord accompanying fracture of the spine, are usually followed by abolition of the reflexes. In the former case the abolition is usually only on the paralyzed side; and in both and all cases, if the patient survive, the reflexes usually return and gradually become exaggerated. Bastian recently called renewed attention to the fact that total transverse lesions of the cord, even far above the seat of the reflexes in the lumbar region, abolish these reflexes. Bruns has also reported a case. I have also recently seen a case of

comminuted fracture of the sixth cervical vertebra, with extensive destruction of the cervical cord, in which the patellar reflexes were abolished. This case is reported and illustrated in my chapter on

FIG. 8.—Getting the Knee Jerk by Re-enforcement. (Dana.)

fractures of the spine, etc., in the "American Text-book of Nervous Diseases," edited by Dercum.

From all these considerations it is seen that there are a few well-

established facts about the reflexes and about their inhibition and exaggeration, but that no theory as yet is quite satisfactory to explain these varying modes.

The principal deep or tendon reflexes are as follows:

The patellar reflex or knee jerk. This is the familiar phenomenon seen on striking or tapping the tendon of the patella while it is kept slightly stretched. When this is done the quadriceps extensor muscle contracts promptly and with considerable vigor, and throws the foot and leg forward in the motion of kicking. To secure this reflex satisfactorily for accurate observation, the patient should be blindfolded and seated on a chair with one leg crossed over the other; or, if the patient is very stout and short-legged, the leg may be supported by the hand and wrist of the observer, passed under the knee and braced upon the patient's other leg; or, best plan of all, the patient may be made to sit upon the edge of a table or edge of a hard bed, with his feet not touching the floor. The tendon is then tapped, and the extent of excursion of the foot is noted. Reinforcement may be used if desired (Fig. 8), and a gauge to measure the range of the foot has even been invented. This instrument, of which there are several varieties, seems to be demanded by an excessive zeal for refinement and precision. The best is probably Lombard's.* The knee jerk is not the same in all persons; it varies widely within the limits of health, and is occasionally even entirely absent in persons apparently perfectly well. Beyond a certain limit, which, however, it is difficult to fix arbitrarily, its exaggeration is always pathological.

Next in importance is the ankle clonus. This consists in a series of clonic contractions in the sural muscles attached to the tendo Achillis, and is excited by putting this tendon suddenly on the stretch. This is done by taking the end of the patient's foot (toes and ball of the foot) in one hand, while the leg is supported by placing the other hand under the calf or ankle (Fig. 9). The foot is then suddenly flexed upon the leg (physiologically extended), the tendon being thus suddenly stretched. The reaction then occurs, the end of the foot (still held in the observer's hand and used as a guide for observation) being jerked in a clonic and rhythmic manner. The excursion, rate, and duration of these clonic movements vary widely, according to case and circumstances. In some cases they are obtained with difficulty; in others they occur with greatest readiness, and the rate is rapid and the duration almost as long as the foot is held in the hand in such a position as slightly to stretch the tendon. Ankle clonus, unlike the knee jerk, is very rarely observed in health, although the statement that it is never thus observed is perhaps too absolute.

The elbow jerk is excited by putting the tendon of the triceps muscle on the stretch and tapping it. This causes a brisk movement of extension of the arm. This reflex is not quite so conspicuous or so readily obtained in health as is the knee jerk.

The wrist jerk consists in a movement of extension of the hand at the wrist when the tendons of the extensor muscles are tapped, while they are put on the stretch by allowing the hand to drop at the wrist (in the position of lead palsy). The middle finger usually reacts

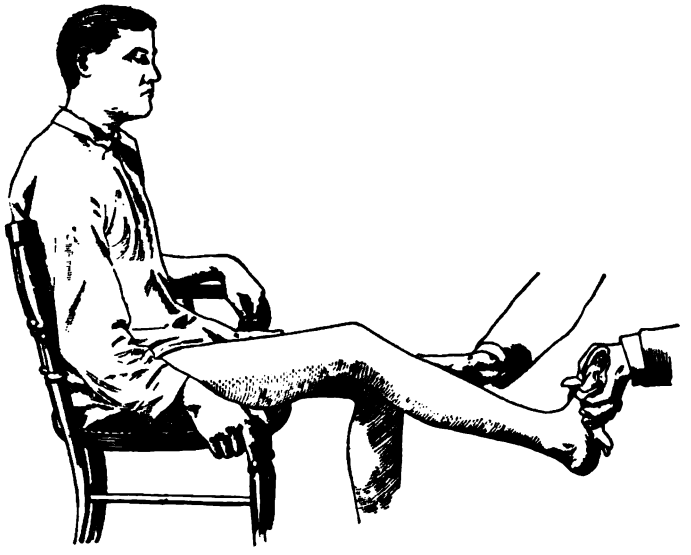

FIG. 9.—Mode of Eliciting Ankle Clonus. (Dana.)

more vigorously than do the others. This reflex is not often obtainable in health.

The jaw jerk or chin jerk consists in a slight reflex action in the masseter muscles when the lower jaw is allowed to hang sufficiently to put the tendons of these muscles on the stretch, and the chin is then smartly tapped. It is not seen in health and is difficult to obtain even in disease.

In cases of some diseases all the deep reflexes of the body are easily excited, so that it is possible to obtain many others than those here mentioned. In fact, a reflex can then be obtained in almost any large muscle by putting its tendon on the stretch and tapping it.

The "paradoxical reflex" is in a sense the reverse of the ordinary tendon reflex. It is caused by suddenly shortening the tendon. It is seen sometimes in the anterior tibial muscle, when the muscle is

suddenly relaxed by flexing (physiologically extending) the foot upon the leg. It is always pathological, and is rare.

The laws of the order of occurrence of reflex action, as stated by Pflüger, are as follows: 1. The reflex occurs on the same side as that on which the irritant is applied, and in muscles whose motor nerves arise from the same segment of the cord; 2. If the reflex occurs on the other side, only the corresponding muscles contract; 3. If the reflexes are unequal on the two sides, then the stronger are on the side of the irritation; 4. If the reflexes extend to other segments, the direction of the extension is towards the oblongata; 5. Finally, all the muscles of the body may give reflexes.'

GENERAL PATHOLOGY.

As already explained, when an axis cylinder or any portion of the neuron becomes separated from that part of the cell body that contains the nucleus, it undergoes a process of gradual degeneration and ultimately dies. There is apparently every evidence that this is a universal law; in other words, that the separated part, say axis cylinder, has no power of maintaining its nutrition permanently by absorbing plasma from the surrounding tisssues in which it lies. This degeneration and death are a comparatively rapid process, leading to a complete destruction and disappearance of the axis cylinder. In spite of this fact, however, the nerve often retains a power of regeneration for quite a long period, and these two processes of degeneration and regeneration, with their histological appearances, must be clearly understood.

It is positively known that this process of degeneration begins promptly when by any influence the axis cylinder is separated from its centre. The process by which it is thus separated may be either by trauma, as wounding, bruising, or cutting, or by disease, especially inflammation and pressure.

As the whole panorama of changes is best and most completely shown after section of the nerve, this immediate description will refer to the changes that are thus produced. Waller, about 1862, demonstrated that this degeneration followed a fixed course in both the anterior and posterior spinal nerve roots; and this is because, as has been already explained, the portion of the neuron cut off from its nutritive centre, the nucleus, is that portion that degenerates. This is the basis of the Wallerian law. It is unimportant in what direction the neuron normally conveys its impulses—it always degenerates in the direction *away* from its cell body. Thus in the ordinary mixed nerve, containing both sensory and motor fibres, which of

course convey impulses normally in opposite directions, the degeneration is always in the *peripheral* portion for both sets of fibres if the nerve has been cut below the junction of the two roots. This is for the obvious reason that in such a case both the efferent and afferent fibres are cut off from their cell bodies. In such a section of a nerve, it is customary to speak of the central end as the *proximal*, and of the peripheral end as the *distal* end.

When a nerve is cut, *i.e.*, when an axis cylinder is severed from the body of the neuron, the following changes occur: According to Ranvier, the nucleus of the sheath of Schwan is the first to show a change. It swells, and the accumulation of protoplasm at this point makes pressure upon the medullary sheath and the axis cylinder within. This swelling occurs, of course, in each of Ranvier's nodes. It soon causes a complete division of the axis cylinder in each of these nodes. The myelin at the same time begins to break up and to form small masses or drops. The sheath grows and its nuclei increase in number. The myelin is still further reduced in amount, probably by absorption. The fragments of axis cylinders shrivel up, and, according to Bowlby,[10] disappear or are difficult to recognize in the course of a short time. Fatty degeneration of some of the adventitious material (nuclei and sheath of Schwann) occurs, and in the *débris* white cells are found, probably acting as scavengers to carry off the products of waste.

According to Mitchell,[11] this process of degeneration begins in the whole of the severed portion of the nerve at the same time, and not, as some have described, at the distal cut end first, then gradually extending from node to node towards the periphery. The same author describes the segmentation of the medullary substance and its final disappearance.

Seegard (quoted by Bowlby) severed the axis cylinder and medullary sheaths by ligating the nerve and quickly removing the ligature. By this process he claimed that the sheath of Schwann was not severed (a doubtful claim). He found practically the same changes that have been described by others. The axis cylinders broke up in places and finally disappeared; the medullary substance ran together in small masses; the nuclei in the sheaths enlarged and segmented, and this process of degeneration attacked the whole of the severed portion of the nerve at once.

The *time* involved in these changes has been carefully noted, and the knowledge on this point is of great clinical importance. Ranvier said that the initial changes (the swelling of the nuclei in the sheath) began within twenty-four hours; hence it is probable, if they are visible within such a short period, that they begin as soon as the

section is made. By the third day there is complete breaking up of the axis cylinder in each node. This, of course, is both physiologically and clinically the most important event in the series. It is the destruction of the essential part of the nerve fibre—the continuation, that is, of the neuron. It is to be noted that this occurs at an early stage of the degenerative process. By the twentieth day the degenerative process appears to be complete. In a human nerve examined by Bowlby on the fifteenth day after division, there were found the same breaking up of the myelin and segmentation of the nuclei, while the axis cylinders were so changed that it was difficult to recognize them. It was thought that fragments of them were present, but at any rate the important fact is that even at this comparatively early day they were practically so broken up as to be recognizable only with difficulty. In a second case examined on the thirtieth day after section, there remained but a few drops of myelin, the axis cylinders were gone, and the nuclei of the sheath were again about normal. From these observations it appears that, in total section of the nerve, the destruction and disappearance of the axis cylinder is one of the earliest results.

From these observations upon sectioned nerves it is not permissible, of course, to draw identical conclusions as to nerves which suffer from other lesions; some qualifications must be made. Thus it is not possible to say in case of localized inflammation, or gradually increasing pressure, or injury from bruising or other wounding, that the changes will always be so prompt and complete as after section. It is certain, however, that the changes are the same in kind, but only differ in degree and in time from those already described. It is especially important in the clinic that this distinction be observed, because the degenerative changes taking place in nerves after disease and even after trauma are not always so rapid and unmistakable as those just described as due to complete section. This, of course, is easily understood when the difference in degree of severity and completeness of the several lesions is considered.

It is necessary to consider especially the effects of *pressure*. This condition may be caused by trauma, by inflammation, or by a new growth. Of course, it is not always or often possible to examine a compressed nerve, because the conditions causing it are usually not permanent. When they are permanent or last a long time, the injury to the nerve also becomes permanent; that is, its axis cylinder becomes divided and the peripheral portion undergoes degeneration, just as from any other grave destructive lesion. In the instances, however, in which pressure is only temporary, the consequent effects also are but temporary; but yet these effects are sufficiently impor-

tant to be noted. Waller, Bastian, and Vulpian experimented on nerves and found that pressure caused a definite train of symptoms—formication, sense of warmth, hyperæsthesia followed by anæsthesia, cramps, palsy. These symptoms observe definite stages, somewhat in the order here given, and disappear also by stages after the pressure is removed." Weir Mitchell also experimented on nerves, causing pressure with a tube of glass containing mercury on the sciatic nerve of a rabbit. He found that the power of the nerve to transmit impulses, when excited by the galvanic current, was gradually abolished. The power gradually returned after the pressure was removed. His conclusion was that the pressure caused mechanical disturbance of the tubal contents; and this conclusion is no doubt the correct one. His examination of the nerve confirmed this conclusion; there was evidence of congestion and disturbance of the contents of the nerve tubes. It is readily conceivable that the delicate cytoplasm of the axis cylinder is thus not destroyed, but merely mechanically compressed and displaced, and that when the pressure is removed it resumes its normal form and constitution. In long-continued and severe pressure there is probably, as already said, a total disruption of the axis cylinder; in other words, a permanent lesion is produced, just as by section, and then the distal portion of the axis cylinder degenerates.

In *bruising* and *contusion* of nerves there is probably a variety of lesions. Some axis cylinders are probably completely divided; others again are practically in the condition caused by temporary pressure, just described. As repair takes place, pressure may also be exerted by proliferating connective tissue, or even by true inflammation, provided the bruised tissues become the seat of infection. Hence in cases of bruising and contusion the symptoms vary according to the extent and severity of the injury. There may be only temporary abolition of function, or there may be secondary degeneration. Thus in the same nerve some axis cylinders may soon recover completely, while others may for a long time continue degenerated.

Closely allied to the subject of degeneration is that of *regeneration* of the divided nerve. Unfortunately much obscurity hangs over this subject, especially because former investigators were not familiar with the neuron as we now understand it. Among these investigators were Tent, Hjelt, Neumann, Ranvier, and Bowlby. The value to be attached to their conclusions may be judged from the fact that all of them, except Ranvier, described the regeneration as taking place by the formation of the new axis cylinder from the nuclei of the sheath of Schwann; and Bowlby even maintains that this regeneration can take place in the divided nerve without union with the proxi-

mal end, and after the first complete degeneration of this axis cylinder already described. Bowlby, in his valuable treatise, maintains this view at considerable length, and even gives an illustration of a longitudinal section of a divided nerve, intended to show the alleged early formation of new nerve fibres from the nuclei of the sheath. The illustration, however, does not show this satisfactorily to critical inspection. If, as Bowlby claims, he found nerve fibres in the distal portion of divided nerves as late as seven and nine months after division, the only conclusion can be that they were preserved intact from the time of section—in other words, that they did not degenerate after section. This is highly incredible. As Bowlby himself acknowledges that similar changes have not been noted by others, it is evident that his observation cannot be accepted as conclusive. It is very evident from our knowledge of the neuron, which we have already described as a complete and individual organism without continuity of structure with any other tissue or substance, that such a process cannot be well conceived. We must apparently wait for new light on the subject from investigators who are familiar with the methods of the new histology. Of those quoted above, Ranvier alone has probably indicated the true process. He states that regeneration takes place only after union of the divided ends, and then by a process of growth downwards or budding of the axis cylinder from the proximal into the distal portion. According to this, there is no true union of the proximal with the distal portion of the axis cylinder. The latter, as already described, has perished and disappeared. But after union of the connective tissue, etc., of the divided ends of the nerve, the axis cylinders in the proximal end, which of course are still in union with the central body, begin to grow into the lower or distal portion of the nerve. This is perfectly in accord with what we know of the power of the axis cylinder in embryonal life; and as this process of repair is essentially the display of an embryonal faculty of growth, there is nothing difficult to conceive in such a process.[18]

The reunion of nerves after section has given rise to much controversy as to the significance of some apparent facts. Glück's experiments on the fowl and the monkey were formerly considered conclusive. According to them the cut ends of a nerve, left *in situ* but unsutured, usually unite in the course of a few days. If a portion of the nerve is exsected the union requires more time in proportion to the length of nerve removed; the removal of one or two centimetres prevents spontaneous reunion, *i.e.*, the ends will unite only if sutured (quoted by Bowlby). If the freshly cut ends are sutured, rapid reunion is obtained; even in eight days "the cut ends were united

by naked axis cylinders." It is even claimed that in the case of a fowl, when complete section of the sciatic nerve had been made, function was perfectly restored in seventy hours. Glück's results, if accepted, point to the fact that primary union, or union by first intention, does occur in nerves. To reconcile these alleged facts with the demonstrable facts of histology is not easy; and they are not in accord, as we shall see, with more recent investigations. With what we know now of the neuron, if we accept as possible the reunion of axis cylinders within seventy hours, and complete restoration of their functions, it would seem almost necessary to believe that each of these minute fibres hunted out and became united with its own particular fragment. If this is not the case, and the reunion is promiscuous, then we must suppose that in spite of their various anatomical distributions, the once divided fibres, after reunion, can take on vicarious functions. Cornil and Ranvier say that "in animals division or resection of a nerve is never followed by immediate union. Therefore it may be doubted if it ever takes place in man." Again, they say that after several months "there is a union of the two ends of the nerves by a process which is not included in any of the methods admitted by surgeons. It is neither immediate nor secondary union, but a special histological evolution, which has not yet been definitely determined." This is evidently the process of budding of the axis cylinders, already referred to, claimed by Ranvier to occur.

Bowlby, whose theory of regeneration of the axis cylinder from the sheath of Schwan has been stated above, of course finds no difficulty in accepting Glück's conclusions, and draws further conclusions from a few surgical cases in his own and others' experiences. In one case, in a man, he saw apparent primary union of the median nerve, with restoration of *sensation*, in eight days; and yet, significant fact, the muscles supplied by the nerve had not regained their reaction to either faradism or galvanism when stimulated through the nerve seven months later. This, to our mind, throws great doubt upon the fact of regeneration, although Bowlby apparently attaches no significance to it. When we consider the free arborizations of the sensory neuron in the skin, it is not difficult to suppose that sensation might be restored by way of another nerve trunk; but when we find the electrotonus of the nerve and muscle not restored after seven months, it is difficult to believe that the neurons are intact from their centre to the periphery. As to the late cases of secondary reunion after suturing, narrated by Bowlby, there is no difficulty in accepting them if we believe with Ranvier that in such cases regeneration begins in the proximal end, and that the axis cylinders, still attached

to their cell bodies, gradually grow downwards from the proximal into the distal portion. The early cases of primary reunion require still further careful observation to clear away some obscurity and some apparent conflict between the facts of histology and the alleged facts of surgery. The facts of surgery and of experiment must certainly conform to the facts of histology before they can be accepted. Bowlby speaks rather slightingly of some physiologists who are unable to reconcile the facts of nerve suture with "their theories as to how injured nerves ought to behave." But the neuron is not a theory; it is an anatomical reality. In cases of secondary reunion without suturing, in which the cut ends of the nerve gradually grow together, a long time is required (as much as a year or eighteen months), and the process is apparently one of downward growth of the axis cylinders from the proximal into the distal portion. According to Bowlby, good results under these circumstances are rare; and this very fact that they are rare points to the conclusion that regeneration, in the sense in which he uses the term, in a thoroughly degenerated nerve is impossible. The apparent exceptions are probably in cases in which the axis cylinder from the proximal portion has succeeded in growing downwards through the distal portion.

The most recent and by far the most conclusive study of regeneration has been made by Stroebe.[14] He used compression, continued for several hours, upon a nerve in a rabbit's ear. In the first day he found the axis cylinder broken, but the sheath of Schwann preserved. There was enlargement of both ends of the nerve, caused evidently by the contents of the nerve being forced out of the compressed portion. Stroebe showed that the new fibres do not arise in the distal portion from the sheath of Schwann, but that these young fibres develop from the nerve stump, *i.e.*, from the proximal end, by a budding process. This proximal end of the axis cylinder undergoes degeneration for the distance of several nodes of Ranvier; this degenerated portion swells, and the new and very delicate fibres spring from the sound portion just behind the swollen end. Several of these may spring from one old axis cylinder. They tend to grow towards the periphery and to force themselves, as it were, through the region to which the pressure had been applied. This region becomes slightly swollen, and is composed of empty sheaths of Schwann, the nuclei of which are proliferating freely. The young fibres possess from the very first delicate medullary sheaths; they pass through the old sheaths of Schwann as far as the area of compression. In passing farther through this region of compression, they seem to follow paths of least resistance, sometimes keeping within the old sheaths of Schwann, sometimes penetrating between them. The distal end of the

nerve is entirely passive in this whole process of regeneration, acting simply as a conducting tube. Stroebe saw the commencement of this process of regeneration on the sixth day after section of the sciatic nerve, and on the seventh day after compression of the nerve of the rabbit's ear. From his description and illustrations it appears plain that this budding of the proximal ends of the divided axis cylinders is an embryonal process of repair, and that these new delicate fibres, springing from the neuron, follow merely a blind impulse of growth through paths of least resistance, and therefore that they can reach the peripheral organs to which the old axis cylinder went, only in case the way through the injured and degenerated nerve trunk continues pervious to them. It may well be conceived that in many cases of grave injury to the nerve trunk this path might be hopelessly blocked, as, for instance, by scar tissue. It is readily seen that Stroebe's work is a confirmation of the ideas of Ranvier.

In a recent paper Ziegler[18] claims that he has demonstrated that the theory of an outgrowth of the new axis cylinder from the stump of the old one is not according to facts. His view is that the old axis cylinder is passive. The active agent in regeneration is a nuclei-containing protoplasm, which is an outgrowth of the sheath of Schwann. From this protoplasm a primary protoplasmic fibre is developed, in which, by a process of differentiation, arises not only a new axis cylinder, but also a new sheath of Schwann and medullary substance. This new axis cylinder is ultimately joined to the stump of the old one. This view of Ziegler's is a recurrence to the old opinion that the new nerve fibre is an outgrowth of the sheath of Schwann, and it is so radically at variance with the opinions now held by histologists, notably Stroebe, that it cannot be accepted until confirmed by recognized experts. It is noteworthy that this view is advanced by a surgeon in a surgical journal.

The *proximal* end of a cut nerve undergoes but slight changes, and these are confined to but a small extent of the nerve trunk. According to Ranvier, only one or two nodes or segments are affected. In these the medullary substance is broken up into rather a finer substance than in the distal portion. The axis cylinder in these affected nodes swells. The nuclei of the sheath of Schwann grow, and large leucocytes from the blood penetrate into the nerve substance.

These changes are probably preliminary to the formation of the *bulbs* which form on the proximal or central ends. These bulbs are composed largely of fibrous tissue. Bowlby says that there are also newly formed axis cylinders in them, and he appears to believe that there is a gradual transition from the newly formed nervous tissue to connective tissue—an opinion, however, which is not tenable. The

bulb is apparently merely the result of a proliferation of connective tissue, which occurs in the effort which nature makes at repair.

Starr[6] suggests that the conflicting opinions about degeneration and regeneration may be due to the fact that the conditions are not always the same. This fact has been already indicated above—the conditions in a freshly severed nerve are different from those in a completely degenerated nerve. This possibility of a difference in conditions is to be noted especially in cases in which degeneration in nerves occurs after injuries other than complete section. In such lesions as are produced, for instance, by pressure, bruising, or even inflammation, the severance of the axis cylinder in every instance may not be complete—in other words, a partial nutrition may be maintained in it. Even in cases in which severance of the axis cylinder is complete, if the nerve trunk itself is not divided, the process of regeneration, by growth downwards of the axis cylinder along the old paths, must be more easily accomplished than in cases in which the nerve has been cut entirely through and then reunited. In cases, again, in which there is complete abolition of function, both motor and sensory, as in inflammation, it does not follow necessarily that there is always complete degeneration. In other words, a partial or perverted nutrition may be maintained. It is evident, therefore, that in neuropathology these two phenomena, *i.e.*, abolition of function and degeneration, should not be confused. Starr[6] gives an excellent digest of the literature of this subject, based especially on the observations of Neumann, Meyer, and Ranvier. As already said, however, we need now the revelations of the newer stains to enable us to trace the regenerated axis cylinders.

S. Weir Mitchell[11] called attention to this fact, *i.e.*, that various degrees and stages of degeneration and regeneration may be occurring in the same nerve in cases of neuritis following injury. This observer also believed, like Bowlby, in the autogenetic restoration of the distal portion of a divided nerve, and said that reparation may occur even when a nerve has been transplanted and grafted upon foreign tissues. But, as already said, Ranvier's explanation of a downward growth of the axis cylinder is more consistent with the facts of histology and our recent knowledge of the neuron.

A still more important pathological process in nerves is *inflammation*. This differs from degeneration in being a more active process. It is always the result of the injurious action of some external agent upon the tissue of the nerve. This external agent is usually, or perhaps always, a poison of some kind. Thus it may be one of the ordinary well-known poisons, such as lead or alcohol; or, on the other hand, one of the so-called toxins that are generated by infec-

tious agents, such as the various bacteria. In cases of nerve injury, in which often many symptoms of neuritis occur, a question may arise whether there is really a true inflammation present; whether there is not rather a degenerative process, such as has been already described, associated with an attempt by nature at repair, which may lead to some proliferation of connective tissue. Such cases were described by Weir Mitchell as results of gunshot wounds. In cases of injury to nerves in which a true inflammation occurs, this is probably due to a secondary infection, *i.e.*, an infection that has taken place in the bruised and disabled tissue of the nerve.

It is customary to describe two types of neuritis: the parenchymatous and the interstitial. These two types blend, and in fact it may be questioned whether they are anything more than different stages or phases of the same process.

In parenchymatous neuritis the first thing attacked is the axis cylinder. This soon swells, but eventually shrinks, and in very severe cases may disappear entirely, just as it does in degeneration. This process is accompanied by changes in the coats of the nerve fibre; the medullary matter is broken up and the nuclei of the sheath of Schwann swell and proliferate. Thus far the process is much like acute degeneration. But in inflammation the minute blood-vessels are involved; their coats are thickened, they are packed with cells, and leucocytes are found wandering in the tissues. It is not often, however, that such a purely parenchymatous type of neuritis is found. It is the type that is seen in some acute cases of neuritis due to the various poisons—alcohol, arsenic, etc.

The gradations from this to the various stages of interstitial inflammation are not difficult to recognize. In many so-called parenchymatous types, in fact, some involvement of the connective tissue is often discernible. This proliferates, and the nerve is consequently somewhat swollen and soft at first. This stage corresponds to the period of acute pains. In more advanced (pure) cases of interstitial neuritis the increase in the connective tissue is conspicuous. This connective tissue between the bundles of nerve fibres undergoes rapid proliferation; its fibres, interlacing with each other, encroach upon and squeeze the bundles of nerve fibres. This increase is especially seen about blood-vessels, the new connective tissue forming concentric rings (see Fig. 10), which gradually press upon and may obliterate the vessel. In the proliferating connective tissue the nuclei stand out very plainly.

The blood-vessels in this form are conspicuously enlarged; they are swollen with accumulated corpuscles, and their coats are thickened. There is also seen extravasation of these cell elements into

the inflamed tissue. Leucocytes abound, and there may be minute hemorrhages, marked by groups of the red blood corpuscles.

Although there is some extravasation of leucocytes and sometimes minute hemorrhages, there is seldom seen true suppuration within the nerve sheath, even in cases in which the nerve has been lying in suppurating tissues and even bathed in pus (Gowers). This is not an absolute rule, however, as in some of the larger nerve trunks, as the sciatic, there may be an acute inflammation with pus. In one very rare opportunity I saw such a condition in a patient who had been suffering with sciatica, and who died after a brief illness from

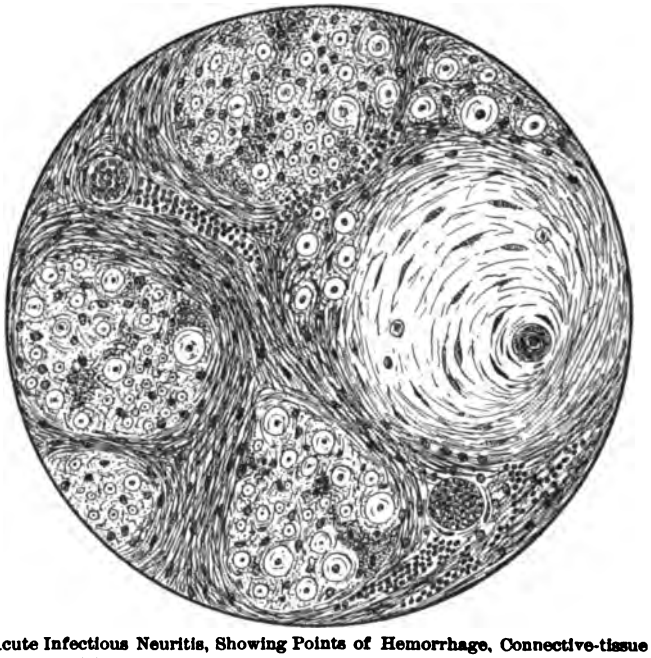

FIG. 10.—Acute Infectious Neuritis, Showing Points of Hemorrhage, Connective-tissue Proliferation, Diseased Nerve Fibres, and Obliterated Vessels. (Rosenheim.)

another affection. In acute inflammation there is usually at least considerable extravasation of serum. Of course, any wandering leucocyte is in truth a pus element; the most that is intended here is that these bodies do not as a rule accumulate sufficiently to form distinct suppuration.

The amount of damage done by the inflammatory process depends upon various factors. The more active, extensive, and long continued this process is, the greater and more permanent will be the injury. First, pressure upon, and then destruction of, the axis cylinder are the essential damages. In cases in which the extravasation, swelling, and proliferation are slight and confined to the connective tissue, the

nerve fibres and their axis cylinders may be but little injured. Pressure may interfere with their function in transmitting impulses, but may not destroy them. In some cases, some axis cylinders are injured and others not. This accounts for many clinical facts. When inflammation is due to the action of virulent poisons, as for instance diphtheria, it is just possible that the injury is not due entirely to the mechanical effects (pressure, etc.), but to direct sedative or toxic action upon the cytoplasm of the neuron. This may account for the fact that sometimes in the so-called parenchymatous type of inflammation the injury done to the nerve structure does not appear adequate for the symptoms produced. In such cases abolition of function in the neuron is an early effect of the infectious process, and is not necessarily in proportion to the amount of reaction of the connective or mesoblastic tissues to this process. As the invasion continues, however, not only the axis cylinder but the blood-vessels and connective tissue begin visibly to show the effect; then the axis cylinder breaks down and degenerates, even without very extensive interstitial changes. At a later stage still, the connective tissue proliferates more and more.

That the neuron is capable of being influenced directly and quickly by various substances is well known, and the fact supports the theory just advanced. Thus acids and even simple salt act as irritants. There is no reason why toxins, generated by microbic action, should not have in some instances a deleterious effect, abolishing function just as a narcotic drug does.

When inflammation results in destruction, even in a limited area, of the axis cylinder, it acts of course as a focal lesion; the distal portion of the axis cylinder, being cut off from its cell body, degenerates, just as it does after complete section of the nerve. It is important to recall this fact when considering the prognosis of inflammation. If the disease has been of long standing and extensive, the resulting degeneration may be as complete as after section, and consequently the process of repair (regeneration) may be long continued.

Inflammation of nerves may be localized or diffused. It may be limited, in other words, to a comparatively few segments, or it may extend through the greater extent of the nerve trunk. The type in this respect varies according to circumstances, especially cause and location. Thus the seventh nerve is apt to suffer from a sharply defined focus of inflammation where it emerges from the bone, while in the type caused by lead the inflammation is extended through more of the nerve trunk. Again, the process may be peripheral and not extend so far as the nerve roots, as is frequently seen in alcoholic cases; in these cases, too, it is not confined to one, but is

distributed to many nerves. An ascending or migrating type is described, in which the disease process may even pass into the cord; this is extremely rare.

Special forms of neuritis are described, as the syphilitic, the tuberculous, the leprous, the cancerous, etc. These depend, for any distinctive marks, upon the special action of the individual poison. A syphilitic neuritis is a common form in some of the cranial nerves —the third and sixth. It is marked by the exudation of a gummatous material, which may even be distinguished in the nerve trunk. Frequently the disease process begins in the membrane of the brain, and involves the nerve secondarily or by contiguity. Tuberculous neuritis is also most common in the cranial nerves in cases of tuberculous meningitis. The leprous neuritis constitutes one of the best known forms of leprosy, the anæsthetic form; in it the inflammatory process is undoubtedly due to a distinct microbe, which has been isolated and described. In cancer the nerve trunks in the immediate vicinity of the growth may be inflamed, and may even exhibit in rare instances the characteristic neoplastic tissue within their sheaths (Cornil and Ranvier).

A neuritis, especially of the parenchymatous or degenerative form, is sometimes seen as a complication of other diseases. In locomotor ataxia, for instance, such a neuritis occurs. In some instances it is more like a degeneration than an inflammation of the axis cylinder, and yet it is probably an active rather than a passive disease process. As suggested above, such a process may be merely the direct action of a toxin upon the sensitive cytoplasm of the axis cylinder. In tabes, moreover, the initial action may be upon the cell body of the neuron in the ganglion of the posterior root; in such a case the axis cylinder degenerates because of the poisoning of its nutritive centre—the nucleus. The painful crises of locomotor ataxia may possibly be caused by the irritative action of a toxin upon the neurons in the intervertebral ganglia. The inflammatory process, under these circumstances, is merely the reaction of the vascular and connective tissue to the irritant, which may be diffused through the cytoplasm of the neuron, as well as carried in the blood.

Some diseases appear to exercise a selective action and affect some neurons more than others. Thus diabetes is occasionally complicated with a neuritis, in which the sensory neurons appear to suffer more than the motor. This causes the so-called *ataxic* type of neuritis, of which more will be said hereafter. It is occasionally caused also by lead and alcohol."

A peculiar disease has been described by Morvan, and is called by his name; it probably depends upon a neuritis. In the early stages

pain is felt in the affected nerves; later, anæsthesia, analgesia, and loss of temperature sense occur, but the characteristic lesion is a painless whitlow, which may terminate in the complete loss of one or more of the distal phalanges of the fingers. Muscular atrophy also occurs. The classical symptoms of syringomyelia are simulated in some degree in these cases. The neuritis is probably due to an infectious process, as Morvan noted a comparatively large number of cases among a fishing community in a town in Brittany. Some observers have claimed that the poison of leprosy is responsible for this train of symptoms; and the neuritis of leprosy does undoubtedly cause severe trophic lesions. Some confusion exists as to the identity of Morvan's disease. Bernhardt, Roth, Broca, and others inclined to the belief that it was the same as syringomyelia. Charcot, Déjerine, and others thought the two were distinct. Morvan claimed that the whitlow was more common than was syringomyelia, and that tactile sense was involved as well as the sense for pain and temperature (Bruhl[**]). The disease, as described by Morvan, has some striking resemblances to anæsthetic leprosy.

Chronic rheumatism, paralysis agitans, and senility are all associated sometimes with alterations in the nerve trunks, which are of either a degenerative or an inflammatory type, but this type is not especially distinct from neuritis as seen in other circumstances.

Finally, morbid growths, *neuromata*, may occur in nerves. They have been distinguished as two forms—true neuromata, in which the growth is apparently formed largely of nerve fibres; and false or pseudo-neuromata, in which the new tissue is largely fibrous (Gowers). They may be single or multiple. Myxomata and sarcomata have been seen growing within the nerve sheath. These cannot be regarded as true neuromata, since that term should be restricted to a true overgrowth of nervous tissue. Whether such a true neuroma ever occurs is a doubtful question (see the description of tumors in the sciatic nerve, below). From a glance at the literature it is evident that small tumors in nerves are like tumors elsewhere—essentially a growth of non-specialized tissue. Of course, nerve fibres may be found in them, but these are probably only the original fibres of the nerve trunk which have become involved in the new growth. From what we know now of the neuron it appears extremely improbable that this can proliferate to such a degree as to form a veritable neoplasm. In all these cases there is probably only an overgrowth of connective tissue. Duhring has described a rare and interesting case of what he called *neuroma cutis*, in which numerous small tubercles were observed in the skin of the shoulder and arm. These were evidently connected with the nerves, and were seen under

the microscope to be formed of connective tissue in which the original nerve fibres were embedded. Pain, in paroxysms of great severity, was a prominent symptom.

Gowers describes a variety of neuroma, consisting of interlacing cords, nodular and tortuous, which he calls plexiform neuroma. The disease begins in fœtal life, and is usually seen on some branch of the fifth nerve, as in the orbit or upper eyelid. In this example, as in all others, the new growth appears to be of connective tissue, enclosing the original nerve fibres. The growth is sometimes myxomatous in character, and is evidently an overgrowth of mesoblastic tissue, not of true nerves. It is possible that the nerve fibres are swollen and enlarged in these cases, but this condition is not the same as a *proliferation* of these fibres. Gowers appears inclined to the belief that true neuroma may occur, but he fails to give a satisfactory instance of it. All his references and descriptions make it plain that there was always present an overgrowth of connective tissue. His statement that ganglion cells have been found, though only in two instances, requires confirmation.

GENERAL SYMPTOMATOLOGY.

The general symptoms of diseases of the peripheral nervous system may be described as they refer to motion, sensation, nutrition, the reflexes, and the circulation. All of these symptoms are due simply to alterations in the normal physiological action of the peripheral motor and sensory neurons; hence it is absolutely neeessary to understand what has been written in the preceding pages about the anatomy and physiology of these nervous structures in order to understand these diseases. In order to avoid undue repetition constant reference will be made to these preceding descriptions. It is essential to reflect also that we are concerned here simply and solely with the peripheral nervous system.

Disorders of Motion.

These are among the commonest and most notable of the effects of diseases of the nerves. These disorders are, briefly, spasm, contracture, tremor, fibrillation, incoördination, and paralysis. Some of these, it is true, are practically not seen when the nerve trunk (axis cylinder) is alone affected, but all of them may occur in diseases that affect the cell body of the neuron. It will be necessary to point out this distinction under each heading.

Spasm, for instance, is seldom seen in affections of the nerve trunk, yet it is not unknown. As already shown, it may occur in

one of the stages of pressure (see page 44). Vulpian and others observed this. It may possibly occur in rare instances in the very early irritative stage of neuritis, but this must be an exceedingly rare occurrence. Other irritative lesions do undoubtedly cause spasm in some nerves. Thus irritative lesions of the superior or inferior laryngeal nerve may cause adductor spasm (Herter"). In some forms of spinal meningitis, in which the nerve roots are irritated, spasm or a spastic state of the muscles occurs. This is usually attributed to irritation of the nerve roots, which of course are composed of axis cylinders exclusively, but it may be a question whether the bodies of the neurons (multipolar cells) are not also irritated in some of these cases. It is necessary, moreover, to distinguish a true spastic state, due to irritation of nerve roots, from an excited reflex spasm or increased tonus (see section on the reflexes, page 34). The form of spasm caused by diseases or affections of the nerve trunks is the tonic, in contrast with the clonic form, which is always a symptom of irritation of the brain cortex. This distinction is important. The gray matter of the brain gives rise when irritated to a series of clonic spasms, epileptiform in character, in the related muscles. The irritation in such a case acts first upon the central motor neuron in the cortex of the brain, the impulse being communicated from it to the peripheral motor neuron (multipolar cell in the anterior horn) in the gray matter in the cord. When, however, the axis cylinder, not the cell body, even of the central neuron is irritated (as, for instance, by faradism), the resulting spasm is tonic. The resulting spasm from irritation of the axis cylinder of the peripheral neuron (motor nerve) is also tonic (Franck "). I have had occasion, in a number of instances, to apply the faradic current to the brain cortex in operations upon the human brain, and I have confirmed this observation, to the extent at least that direct irritation of the motor cells in the cortex causes clonic spasms, persisting after the withdrawal of the irritant." Moreover, the clonic spasm, caused by exciting the motor cells in the cortex, persists for some time after the irritant is withdrawn; whereas the tonic spasm, caused by applying the faradic current to the exposed fibres in the white matter in the bottom of the wound, after excising the cortex for epilepsy, ceases as soon as the irritant is withdrawn (Franck, Lloyd). In this latter case, the exposed fibres in the white matter are, of course, axis cylinders.

Painful tonic spasm or cramp is an occasional symptom of multiple neuritis in its early stage. It may also be caused by pressure on a nerve trunk, as is seen not seldom in parturition. In this case, however, it is possible that interference with the circulation may be

a causative factor. The most common seat of cramps is in the muscles of the calf of the leg.

Contracture is a state of persistent contraction or shortening of a muscle. This is the usual explanation, although the condition in all cases is probably not the same thing physiologically as contraction. Contraction is the physiological act of shortening by which the muscle performs its function; it is, as a rule, rather brief in duration, although it may persist for variable periods of time. Contracture, on the other hand, is a persistent state of shortening of the muscle, which as a rule is no longer under the control of the will; and this contracture can be overcome only by passive effort. It is always pathological. By some it is explained as a sort of persistent exaggerated reflex tonus. It occurs, for instance, in cases in which the muscle is cut off from the volitional centres, as in old cases of hemiplegia. This form of contracture is also seen in diseases of nerves. In some forms of neuritis the contractures which occur may be permanent and cannot be overcome readily even by passive forcible movements. These contractures appear to be caused in the less paralyzed muscles, as the flexors, and may be explained by the fact that the extensors are so completely paralyzed as to offer no counterforce to the flexors. They are thus in a sense physiological, *i.e.*, they are due to the natural contractility of the muscular fibre when this is unopposed.

Tremor, which is not a rare symptom in diseases of the central nervous system, is very rare in those of the nerves. It is a rhythmical to-and-fro movement, of rather limited range, due to the alternate contraction and relaxation of opposing muscles. It is no doubt due to alterations in the motor neurons, in those both of the cortex and of the spinal cord. When it occurs as a symptom in diseases of the peripheral nervous system, the likelihood is that the cell body of the neuron is affected, and rarely the axis cylinder only. Tremor is said to occur in some cases of neuritis; in such instances it is possibly caused by the debilitation of the axis cylinders, so that the normal nerve impulses flow both feebly and irregularly. Tremor occurs also in hysteria, neurasthenia, alcoholism, chronic poisoning with lead and mercury, exophthalmic goitre, and the exhaustion caused by acute febrile diseases. The exact seat of the tremor cannot be stated with certainty in most of these diseases, and yet from their nature this seat is not probably in the nerve trunks (axis cylinders) exclusively. It is more probably in the whole of the peripheral motor neuron, since this anatomical structure is a unit and likely to be affected as a whole by poisons and general nervous states.

Fibrillation differs from tremor, among other ways, by the fact

that it is an affection of a few individual fibres rather than of the whole muscle. It is an involuntary movement of these fibres, rather sluggish in character. It is a wave-like motion, seen just beneath the skin, and so feeble that it does not move the muscle as a whole, and of course causes no movement in the parts to which the muscle is attached. It may occur in only a few fibres at long intervals of time, or it may be diffused through many fibres of the muscle, the successive waves of fibrillation chasing each other in rapid succession. In cases in which they do not occur spontaneously or frequently, fibrillary contractions can often be excited by smartly tapping the skin over the muscle with the finger. Exposure of the surface to the cold air, as by removing the bed clothes or body clothes, will often excite them. Fibrillation is probably caused by a lesion which at once weakens and irritates the cell body of the peripheral motor neuron in the anterior horn of the spinal cord. It is not caused by diseases, pure and simple, of the nerve trunks or axis cylinders of the neurons. It is mentioned and described here in order that its negative value as a symptom of the peripheral nervous system may be understood. The cell body in the cord and the axis cylinder in the nerve trunk belong to one and the same anatomical unit, the neuron. It seems, however, that fibrillation is excited only by an irritant, continuously or almost continuously acting upon the cell body, *i.e.*, the multipolar cell in the anterior horn. This symptom, therefore, is almost entirely confined to progressive degenerative processes that affect and gradually destroy the large ganglionic motor cells. Such a disease especially is chronic anterior poliomyelitis. While this is the opinion usually entertained of the significance of fibrillation, there does not seem any *a priori* reason why irritation applied to the axis cylinder or its ramifications should not be able to cause this symptom. Herter[1] expressly states that degenerative neuritis may cause fibrillation, and even intimates that irritation of the motor nerve endings may also possibly cause it. This, however, is as yet a somewhat speculative question.

Incoördination is an irregularity of movement caused by a series of unequal and improperly adjusted muscular contractions. To understand it, the normal association of the muscles in producing complex movements must be considered. In a voluntary movement, depending upon the mutual action of several allied and opposing muscles or groups of muscles, the precision of the movement depends upon the exact relative force exerted by these several groups. This precision, in fact, represents a balance of power so adjusted that the exact movement is the result, and the resultant, therefore, is the effect of the combined action of these muscles. All the muscles involved must

contribute just the proper amount of force, and just at and for the proper duration of time, in order that the movement may be exact. The exact seat of this power of coördination is no doubt in the central nervous system (cerebellum or motor cortex ?), and precision of movement is the result of education. This power depends undoubtedly, however, upon the integrity of the peripheral system, especially the sensory system and the muscular sense. Hence it may be disturbed by a variety of diseases, both central and peripheral. Thus there is a cortical incoördination or ataxia, as well as a cerebellar, a spinal, and a peripheral one. In the forms due to central disease, as sclerotic processes, cerebellar tumors, etc., the central seat of this function is involved, while in the forms due to peripheral disease the nerve trunks or their ends are affected. The muscular sense is no doubt largely the basis of supply, as it were, for the impulses upon which the central organ depends for a proper exercise of this function. In locomotor ataxia the incoördination probably depends upon the affection of the peripheral sensory neurons. Incoordination or ataxia, as a symptom of disease of the peripheral nervous system, is seen especially in some forms of multiple neuritis. In this disease the symptom is probably caused by involvement of the sensory fibres in the skin and in the nerve trunks. Hence anæsthesia is usually a marked feature of these cases. They present an ataxia almost if not quite identical with that of locomotor ataxia, and thus lead to a confusion of these two diseases. Such cases have been described under the title of pseudotabes.[17] This form of multiple neuritis is caused especially by alcohol, lead, and diphtheria. In 1884 Déjerine[18] described this affection under the head of *nervotabes périphérique*. He had observed it in two alcoholic subjects, both of whom had incoördination, anæsthesia, abolition of the knee jerks, atrophy, and paresis, without involvement of the eye muscles or bladder. Post mortem he found inflammation of the cutaneous nerve endings, slighter changes in the intramuscular nerves, and no change whatever in the posterior roots, their ganglia, or the spinal cord. About the same time Dreschfeld [19] described a type of alcoholic ataxia; his clinical picture was strikingly like Déjerine's, but was without such careful post-mortem findings. Kruche[20] also wrote a paper on "Pseudotabes in Alcoholics." Even before these observers, however, Wilks, in his lectures in 1878, and, still earlier, Leudet [21] described the same condition. Leudet wrote before multiple neuritis was generally thought of, and he came near to describing what we recognize to-day as polyneuritis. In 1888 Leyden wrote a monograph, in which he distinguished five forms of multiple neuritis, one of which he named the sensory form and grouped under it the

acute ataxias, whose cases are distinguished especially by incoördination and depend upon the lesions of the peripheral sensory neurons here referred to. These cases depend upon the involvement of the peripheral endings of the cutaneous nerves—the lesion described by Déjerine. In the case described by the writer the patient was a painter who had been exposed to lead for many years, and who also had been a hard drinker. His symptoms so closely resembled tabes that his case had been diagnosticated as such by a competent observer, but the autopsy revealed no involvement of the spinal cord. The importance of recognizing these facts in neuropathology is very great, because if they are ignored a comparatively curable disease may be mistaken for tabes, which is practically incurable.

Paralysis in some form is the most common symptom of disease of the peripheral nervous system. When thus caused it usually has some general features that serve to distinguish it. First, for instance, its distribution is often characteristic. Thus, if it results from a disease limited to one nerve trunk, it is found only in the muscles supplied by this nerve. This form of paralysis is usually so conspicuous and characteristic that mistake is wellnigh impossible. Good examples of it are seen in paralysis of the third and of the seventh nerve. In cases in which more than one nerve is involved the groups of muscles that are paralyzed have often a rather irregular distribution, and are not necessarily physiologically associated as they are likely to be in paralysis from cerebral lesions especially, and to a less extent from spinal lesions also. The degree of paralysis is a second characteristic of some value. The muscles, when supplied by one nerve, are usually equally paralyzed, and in many cases they are absolutely paralyzed. There is no paralysis more complete than that caused by a severe lesion of a nerve. The peripheral motor neurons being in such a case completely disabled for transmitting impulses, the resulting paralysis will be absolute. This rule, of course, is not invariable, because slight lesions may permit some impulses to pass; neither is it distinctive, because absolute paralysis may be caused by lesions of the brain and cord; but it is nevertheless often characteristic when observed in a limited muscle group which is supplied by a single nerve—as in the face, for instance. Third, the kind of paralysis is quite characteristic in cases of peripheral lesions; the palsy is always flaccid, the tone of the muscle is always diminished or lost. Consequently secondary spastic states do not set in, as in the case of hemiplegia and of paraplegia due to a middorsal lesion. In long-standing cases, it is true, a late contracture often sets in, but this is not spastic and does not imply an increased tonus in the muscles. It is not easily explained, but may

be due to the degenerative processes taking place in the tissue of the muscles. In line with these facts is the abolition of the reflexes that have their course through the affected nerve trunks. Peripheral palsy is always characterized by the loss of these reflexes. This of course is inevitable, from the fact that the motor neurons from the cord (and probably the sensory neurons too) are impaired. This flaccid paralysis, with loss of muscular tone and of reflexes, being caused by disease or injury of the peripheral motor neuron in any of its parts, is seen as well in those diseases that affect exclusively the cell body of this neuron in the spinal cord (as anterior poliomyelitis), as in diseases of the nerves. Diseases of these two regions can be told apart only by other symptoms than the paralysis. As said already, the degree of paralysis may vary, even in cases of peripheral lesions, although not so commonly with them as with central lesions. A partial or mild degree of paralysis is sometimes called *paresis*, but this distinction refers entirely to the degree of paralysis, not to its cause or kind. Finally the reactions of degeneration, to be described later, occur in this form of paralysis.

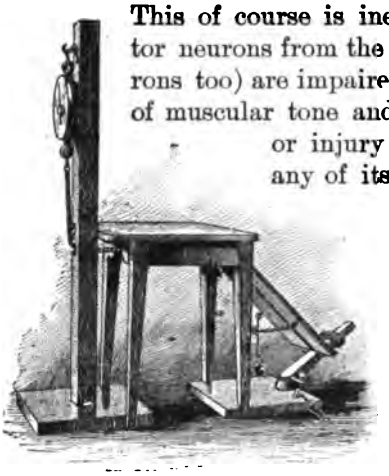

FIG. 11.—Leg Dynamometer. (Dana.)

To test paralysis is usually not difficult; only simple tests are requisite. Some instruments of precision are in use, as the dynamometer, for registering the power in the grip. Dana has designed a leg dynamometer and a foot dynamometer (Figs. 11 and 12). All these instruments merely measure the power of particular groups of muscles, and do not give much more exact knowledge than can be gained without them. The power of the grip, for instance, can be better tested in many cases by the physician giving his hand to the patient and having him squeeze it; in this way an accurate idea of the strength and mobility of the muscles is directly imparted, that cannot be gained by the instrument. Moreover, a comparatively small number of muscles are adapted to precise tests; in most of them simple observation by sight and by resistance is alone possible. To test the power of the legs a few simple methods suffice; thus the physician may flex the patient's leg while

FIG. 12.—Foot Dynamometer. (Dana.)

the latter lies on his back, and, holding the sole of the foot firmly in his hand, request the patient to extend the leg forcibly. In hemiplegia the patient cannot extend his foot against this resistance; even in hemiparesis the effort is in marked contrast with the sound side. In multiple neuritis the patient, as he lies on his back, cannot lift his entire leg from the bed at once (flex it at the hip) without bending the knee. When he attempts to do so, the knee is bent and the heel does not leave the bed, but is dragged up along the surface of the bed towards the buttocks. This is because of the paralysis of the extensors. In unconscious or semiconscious states a paralyzed leg or arm loses all resistive power; hence the sense of resistance is not imparted to the observer as it is by the unparalyzed member. This extreme flaccidity is seen in apoplexy with hemiplegia. In the case of some of the small muscles and groups of muscles certain precautions must be observed. The tongue, for instance, deviates towards the paralyzed side, because physiologically it is pushed out of the mouth; consequently the muscles of the sound side push it over towards the unopposed side; but in some cases in which the face is paralyzed, the corner of the mouth droops, and this may cause an appearance of slight deviation of the tongue, which is deceptive. The extrinsic muscles of the eye require careful observation to determine their paralysis, but this subject will be referred to again in the proper place. Changing the attitude sometimes gives valuable indications of weakness or paralysis of certain muscles; thus in rising from the reclining or sitting to the standing position, the patient may not be able to use certain essential muscles. In some of the forms of muscular dystrophy, as pseudohypertrophic muscular paralysis, the weakness of the muscles of the legs is best shown by the patient's characteristic way of "climbing up his own legs" in rising from a sitting position on the floor. Again, the gait is often characteristic; thus the gait of multiple neuritis is thought to resemble that of a turkey cock, because of the dropping of the foot as the leg is lifted, due to paralysis of the extensors.

Disorders of Sensation.

As already explained, sensation has a variety of modes. These, briefly, are the tactile sense, the sense for heat and cold, the sense for pain, and the muscular sense. Their physiology has already been discussed (page 28). Considerable obscurity clings to the question whether these various modes have distinct paths in the nerve trunks, *i.e.*, whether the sensory neurons are differentiated so as to carry only separate and distinct modes of sensation. Whatever final

answer physiology and histology may give to this question, the clinical fact is plain that these varieties are separable by disease. As the course of all of them is by way of the peripheral sensory neurons from the periphery to the spinal cord, it is evident that all of them may be affected by diseases that affect the integrity of these neurons. Hence among the symptoms of diseases of the peripheral nervous system some alteration in sensation is very common.

The commonest of these varieties is abolition of the *tactile sense*, or tactile anæsthesia. It is commonly seen in all kinds of lesions of nerves, as, for instance, inflammation, pressure, and the various traumata. The patient himself does not always necessarily know that he has tactile anæsthesia; or, on the other hand, some subjective sense of numbness may accompany it. This symptom is readily overlooked by a careless observer; it should be sought for, and the skin minutely tested, in all cases of disorder of the peripheral nervous system. Abolition of sensation is not always coexistent or coextensive with abolition of motion. As a matter of rather common observation, motion may be much more profoundly affected than sensation in lesion of a common nerve trunk. Thus there may be complete paralysis of motion, and yet sensation may be in part preserved. Again, in cases of nerve injury, in which both motion and sensation are abolished and in which recovery occurs, sensation may be restored for quite a period before motion. The explanation of these differences in the action of the two sets of neurons is not very apparent; probably the sensory impulse is more minute and delicate than the motor impulse, and makes its way more readily through the cytoplasm of the cell; or less of an impulse may be required to excite sensation in the cells of the brain cortex than to cause a muscle to respond. If this is the explanation, the difference depends not so much on a difference in the two sets of neurons as on a difference in the excitability of their receptive organs.

To test the tactile sense the patient should be blindfolded. Any pointed instrument may be used, as a pin, a pen, a penknife, or a hairpin. The best, however, is the æthesiometer. This consists of a hinged two-pointed instrument, somewhat resembling a draughtsman's compass, with a scale for measuring the distance of the points from each other. A good plan, which I usually adopt, is to have the patient count aloud the number of times he is touched until the number four is reached; then to begin again, and count again as far as four, and so on. The advantages of this plan are numerous. It permits the observer to detect readily, for instance, an inattentive patient, and, better yet, a malingerer. The former will count by rote, even when he is not touched, and his fault can

thus be corrected; the latter will involuntarily and almost inevitably count when the alleged anæsthetic spot is touched, especially if the process of counting has been going on for some time. On the other hand, the patient with a genuine anæsthetic area will inevitably drop a count when this area is touched in the midst of the count. By this method also the exact limits of the anæsthesia can be mapped out. In addition to this test, it is desirable to know how well the patient can appreciate the distance between the points, and the results can be compared with the table for normal results (page 31). The patient's ability to recognize location should also be noted. The sense of *contact* is, of course, only a degree of tactile sense and does not require special tests. The sense of *pressure* is in part closely allied to the muscular sense. It may be tested with weights of different sizes, laid upon the anæsthetic spot; but these tests are of dubious value, because this faculty varies so much in individuals within normal limits. The same may be said of Rumpf's test by writing. By this test the letters are traced upon the skin with a hard-pointed instrument, and the patient is requested to name them. Rumpf's table for these responses (given by Dana) is probably subject to wide variations. The test is practically of little or no value. Of greater importance is to note whether the tactile sense, if not abolished, is in any degree delayed. In clinical work, in which great precision in determining this point is often not practicable, the observer must be content with an approximate result. It is not difficult, for instance, to determine whether the response is long delayed, although this cannot be measured to the fraction of a second. Dana gives the following table of the normal reaction time:

A touch on the hand, about	0.12 second.
A touch on the foot, about	0.17 "
Hearing	0.13 "
Sight	0.16 "
Taste	0.15 "

The delay, which may amount to several seconds in cases of cerebral lesion, is not likely to be considerable in cases of peripheral lesion.

The *temperature sense*, like the tactile sense, is abolished by any lesion that totally destroys the continuity of the axis cylinder or so interferes with it as to impair its conducting power. Hence it happens that in cases of complete tactile anæsthesia from a peripheral lesion there is always found also anæsthesia to heat and cold. Abolition of temperature sense, without any tactile anæsthesia, is not often observed in peripheral lesions. This dissociation is absolutely denied by some authors, especially for neuritis. Thus Bruhl[*] claims that the peculiar dissociation symptom (*i.e.*, preservation of

tactile sense with abolition of the temperature sense and pain sense), seen in syringomyelia, is never seen in neuritis. I have put on record, however, two cases of injury to the cervical region of the spinal cord in which the nerve roots were probably injured and involved in adventitious material, and in which this dissociation symptom was a marked feature." These cases seemed to prove that spinal pachymeningitis and neuritis may cause loss of temperature sense without anæsthesia; still, they cannot be said to prove this point incontestably, because it was not known to what extent the central gray matter of the cord was injured; if it was injured, this injury would probably account for the anæsthesia to temperature and to pain. In anæsthetic leprosy, in which the lesion is a neuritis, this dissociation of tactile and temperature sense has been noted. In fact, the resemblance of this form of leprosy to syringomyelia is marked. Charcot" has described a case of injury to a dorsal nerve in which this dissociation was seen, and says that others have seen it in peripheral neuritis. If, therefore, neuritis can in some instances cause impairment of temperature sense without tactile anæsthesia, this state would probably indicate that the peripheral sensory neurons are not differentiated for these varieties of sensation. From the clinical standpoint, however, it must be said that, as a rule, the dissociation of the two is not commonly seen in cases of peripheral lesions, but is highly characteristic of lesions of the central gray matter of the spinal cord, as in syringomyelia. It is significant that the temperature sense and pain sense are usually affected together.

To test the temperature sense, two small bottles or test tubes of exactly the same size are used. One is filled with hot, the other with iced water, or, better yet, with small bits of cracked ice. Extremes of heat and cold had best be used. The bottles or tubes should be tightly stoppered, to prevent leakage upon the patient's skin. The patient should be blindfolded, and then touched at haphazard on the areas to be examined. Alternation should be made without system. As a rule, patients will more frequently call cold hot than *vice versa*, especially if the object is very cold, as a bit of ice. Variations of temperature within a few degrees are recognizable by patients in health. Below 60° F. the sensation of cold is imparted, and above 100° F. the sensation of warmth, gradually increasing from that point to a sensation of heat.

The *pain sense* is abolished (analgesia) in complete tactile anæsthesia, as a rule. In other words, a peripheral lesion so complete as totally to obstruct the passage of the tactile impulses will not permit the passage of painful impressions. There are, of course, minor degrees of tactile anæsthesia which are compatible with the passage

of painful impulses; in these cases the neuron is evidently not completely cut off from its terminal arborizations, and, while unable to respond to an ordinary tactile stimulant, can react to a stronger painful one. This condition must not be confused with "anæsthesia dolorosa," in which the pain arises from an irritative but obstructive lesion in the course of the nerve trunk, the pain being referred by the mind to the periphery. Something like this is seen in multiple neuritis, in which disease the nerve trunks are tender and sore, and yet do not always conduct tactile impulses from the periphery to the nerve centres. The pain sense, as already pointed out, is in some way apparently associated with the temperature sense; the two are usually abolished together. This is seen in lesions of the central gray matter of the spinal cord, as syringomyelia, the tactile sense not being affected; but in peripheral nerve affections this dissociation is very rare. Charcot,[27] as already said, saw this dissociation in a case of injury to a dorsal nerve, and others have reported it in the neuritis of anæsthetic leprosy. Gowers[28] claims that he has twice, in multiple neuritis, seen the pain sense lost, the tactile sense being preserved. As a rule, however, in the clinic this dissociation is not seen in cases of disease of the peripheral nervous system. The commoner dissociation is that first described, in which with a mild form of tactile anæsthesia there is some preservation of the pain sense.

To test the sensibility to pain the skin may be pinched. A concealed needle, controlled by a spring, is sometimes used. An instrument, containing such a needle, has been designed and called the *aiguille cachée*. It has the advantage of not exciting the patient's suspicion or apprehension; hence the exact degree of pain sense can be determined, and malingering can be exposed. With the patient blindfolded, however, an ordinary needle can be used, and is quite as good as the instrument. The best test for the pain sense is the faradic current. The strength can be altered at will without the patient's knowledge, and his exact sensitiveness to painful currents can thus be noted.

The opposite to analgesia is hyperalgesia. In this condition ordinary non-painful impressions may become painful. It is evidently caused by an irritative lesion. It is practically the same thing as that condition in a nerve which is called *pain*. Hyperalgesia may be described as a state of increased susceptibility to pain, whereas pain itself is a modification, *sui generis*, of the neuron, caused apparently by over-stimulation or irritation.* In the hyperalgesic neuron it is

* An exact definition of pain is impossible. The subject shades off rapidly into obscure psychological and even metaphysical definitions, as can readily be seen by consulting Mr. Marshall's book already referred to.[4] There is a school of psycholo-

easier to excite pain than in a normal neuron, because it is already in a state of irritation; when this irritation is slightly intensified, pain is the result. That irritation is the cause of pain is evident in some cases from the fact that the nerve trunks are painful on pressure, This is so in neuritis. In such cases the irritation acts on the axis cylinders of the neurons in the nerve trunks. Painful sensations, as burning, are in these cases often felt in the periphery, as in the soles of the feet, but on pressure the nerve trunk itself is also sore. The clinical appearance and significance of pain will be described elsewhere. It may vary in character, being boring, tearing, burning, or aching, according to circumstances.

The *muscular sense*, by which the mind is made familiar with the state of contracture, the efforts, and the position of the muscles, may be affected by disease. As the pathway for this sense from the muscles to the nerve centres is generally believed to be by the sensory neurons, it follows that affections of these neurons should affect the muscular sense. This is the case in several diseases. In locomotor ataxia, in which affection these peripheral sensory neurons are extensively diseased, the muscular sense is often much impaired. In the ataxic form of multiple neuritis, in which disease also these sensory neurons, especially their peripheral endings, are gravely impaired, the muscular sense is often abolished. Doubtless a complete obstructive lesion of any single nerve causes some impairment of the muscular sense in the muscles to which the nerve is distributed, but in lesions of groups of nerves, affecting especially the legs, the disturbance is more conspicuous. That the muscles have sensory nerves is well known; the pain caused by cramp in the muscles is sufficient evidence that the muscle's state of contraction is instantly announced to the consciousness. That the muscular sense is dependent upon these sensory nerves in the muscles themselves, and not upon the sensory nerves in the skin, is apparent from a number of observed facts. Thus there may be anæsthesia of the skin without loss of the muscular sense. According to Landois, a frog deprived of its skin can jump in a normal way. The muscular sense is also no doubt supplemented by the sensibility of the joints, fasciæ, etc. Again, there may be loss of sense of the posture of the limb while the cutaneous sensibility is normal.

The sensory nerves in the muscles are no doubt sensitive to many

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gists, since Kant's time, who deny that pleasure and pain are sensations. Such a debate is largely a question of terms and definitions. There are, of course, psychical pleasure and pain independent of bodily states; but this does not constitute a reason for denying that there are also physical pleasure and pain, dependent entirely upon bodily states.

sorts of impressions, just as are sensory nerves elsewhere. Thus they can convey painful impressions, as of cramps, already referred to, and of inflammation. But this sensibility is not included in the term muscular sense, which simply refers to the contraction, effort, and location of the muscle. It follows, therefore, that a sense of resistance, as well as of effort, is an important element of this sense. As all muscular effort is directed against some resistance (if this is nothing more than gravitation), it follows that in the normal state the mind estimates with some degree of precision the force of the resistance and the consequent force necessary to overcome it. Hence both the sense of pressure and the sense of weight (the latter especially) are important elements in the muscular sense. So also the sense of movement and position are a part of this sense. The sense of fatigue is no doubt due to an impression made upon the sensory nerves in the muscles.

To test the muscular sense weights are generally used. They are suspended from the foot or hand, or placed upon any part of the body to be tested, and the patient then estimates their comparative weight. To do this, especially if the weights are suspended, requires a fine muscular adjustment and expenditure of force, which it is the function of the muscular sense to estimate. These weights should be of uniform size and feeling so that the patient cannot distinguish between them. They may be hollow objects, as balls or cylinders, which can be variously weighted with shot or sand.

Besides the abolition of the various modes of sensation there may occur various modifications of sensation as results of disease. One of the most important of these is pain, which has already been discussed. Another is *parœsthesia*. This is a state of changed or perverted sensibility, and is marked by a variety of modifications of normal sensation. Among these varieties are burning, stinging, and itching; also the sensation of ants creeping on the skin (formication). Some of these approach closely to pain in character. There is no objective method of testing for them; as they are entirely subjective, the evidence of them rests entirely upon the veracity and accuracy of the patient. These parœsthetic phenomena depend probably upon mild forms of irritation of the peripheral sensory neurons somewhere in their course. Hence they are seen in cases of neuritis; also in cases in which the neurons are irritated by foreign or toxic substances in the blood. An instance of the latter is probably seen in some cases of mild poisoning by morphine, in which itching is a disagreeable symptom. In gout, or the lithæmic diathesis, similar symptoms are occasionally observed.

The special senses—sight, hearing, taste, and smell—are also

subject to various changes by disease. They may be abolished or variously modified. These affections will be described under the diseases of their special nerves.

Disorders of Nutrition.

These are among the most common effects of peripheral diseases. They are seen in the muscles, the skin, the subcutaneous connective tissues, the vascular system, the joints, and, to a much less extent, in the bones. The theories which have been advanced to account for these trophic disorders are, none of them, entirely satisfactory. As said already when describing the neuron (page 4), the axis cylinder, or any other part of the nerve cell, will waste and perish when cut off from its trophic centre—the nucleus. This is not difficult to understand, seeing that the neuron is an anatomical unit and that its parts depend for nourishment upon the nucleus. But this rule does not hold good, of course, with other tissues, such as skin and muscle. These tissues, while in direct anatomical relation with the neurons, are not in direct nutritional relation with them, since they derive their nourishment from their own independent vascular supply. In the case of the muscles, for instance, this vascular supply is copious, and so far as can be seen from a study of the terminal arborizations of the motor neurons in the muscles, is not directly influenced by these; yet the muscles are among the first of the various structures to degenerate after severe lesions of the nerves. One theory, supported by Samuel, attempts to explain these changes by assuming the existence of trophic nerves. How these nerves perform their function is not explained; their existence, moreover, is not demonstrable. Duchenne said: "If we had no knowledge of such (*i.e.*, trophic) nerves, we should be forced to invent them"—a feat, by the way, which some physiologists have been nothing loth to attempt. Weir Mitchell, in his classical studies of injuries of nerves, made careful examination of this subject, and decided against the theory of "trophic" nerves, and he regarded with favor the possibility of disorders of nutrition being produced by irritation of ordinary sensory and motor nerves. Another theory relies upon the office of the vasomotor system; but this too is not clear. It seems that all we know is the clinical fact that lesions of the nerves cause nutritive changes and that these are readily recognizable. These lesions may be either irritative or destructive. According to Lewaschew, quoted by Gowers," if a cord is passed through each sciatic nerve and one nerve is further stimulated with irritating fluids, trophic lesions occur more readily in the limb of the nerve thus irritated than in the

other. Destructive lesions, as section or inflammation o. a nerve, are also followed by characteristic changes in nutrition. A rational explanation of these changes, so far as the muscles are concerned at least, is that when this tissue no longer acts it no longer assimilates plasma. We have seen that something like this occurs also in the neuron. In other words, the normal activity of a vital tissue is the only cause that both requires and permits it to use up pabulum. Function and nutrition are in the relation of cause and effect. Function, by consuming plasma, stimulates nutrition; without it there is no demand for new plasma, hence nutrition grows weaker and weaker.

Weir Mitchell's general conclusions on the subject of trophic lesions following nerve injuries are as follows:[11]

Total section of the main nerve trunks of a limb results in atrophy (of all tissues?) but not necessarily in inflammation. But it does not prevent inflammation, nor, on the other hand, repair.

Partial wounds, especially gunshot lesions, give rise to trophic changes in the skin, hair, nails, areolar tissue, and muscles. Except the entire arrest of the growth of the nail, every trophic change that can arise from injury or disease of the centres can also arise from wounds of the nerves.

Section of motor nerves causes atrophy and contraction of the related muscles. Partial wounds of these nerves cause various degrees of wasting and loss of muscular sense, and loss of power to respond to electrical and mechanical irritants.

Mitchell claims also that trophic changes in the skin, hair, and nails are never present without some affection of sensation; but that affections of sensation may exist without trophic changes.

Finally, trophic changes are most prone to follow wounds of nerves which are distributed to the hands and feet, and occur more rarely when the injury involves only the branches to the upper portions of a limb.

Mitchell's statement that every trophic change that can arise from central disease may also arise from injury of the nerves, is a most important one. It may be considered, perhaps, rather too absolute, and yet increasing knowledge of the nervous system (especially its systems of neurons, their relations to each other, and to the peripheral organs) renders it more and more probable that this opinion, formulated thirty years ago, will yet be found to be the correct one. For it is certain that all trophic influence from the nerve centres must be communicated by way of the peripheral neurons, and that this influence cannot be bad if these neurons are themselves perfectly healthy. This is shown, for instance, in the case of the trophic lesions of locomotor ataxia, which is usually considered a central dis-

ease; yet it is *not* a central disease exclusively, or perhaps even primarily and essentially, because it is a disease of the peripheral sensory neurons; hence its trophic lesions are not in reality of central but rather of peripheral origin. It will be necessary, however, in discussing trophic lesions here, to endeavor to make a distinction between those that appear clinically to be of central origin and those that are caused especially by affections of the nerves. These latter alone belong to the present subject.

Trophic Lesions of the Muscles.

The muscles begin to exhibit trophic changes soon after they are cut off from the cell body of the motor neurons in the anterior horns of the spinal cord. Evidence of these changes may be observed in a few days, especially in the electrotonus. Any lesion whatever that acts as a destructive or obstructive one to cut off the nerve influence will cause trophic disturbance in the muscles. Hence all kinds of wounds and contusions, as well as pressure and inflammation, act thus. Brown-Séquard attempted to draw a distinction between the trophic effects of destructive and those of irritative lesions of nerves. Only the latter, according to this observer, were followed by truly degenerative changes in muscles. This theory has now only historic value, and serves to illustrate the excesses of pure physiological speculation (Arnozan[11]). The exact changes in the muscles are not merely such as would be caused by disuse, but indicate a rapid destructive process. Vulpian and others have observed that the muscular fibre breaks down in granules; fatty metamorphosis occurs in these, and they are gradually absorbed. The connective tissue proliferates, and finally the muscular tissue is entirely replaced by a mass of fibrous connective tissue which contracts and presents no elasticity whatever. These changes of course are soon apparent to the eye in the altered contour and flabby appearance of the muscle; as they progress, the body of the muscle diminishes in size; it no longer feels or looks like muscular tissue, and finally is plainly converted into a non-muscular substance.

The most characteristic changes shown in degenerating muscle are the alterations in the normal electrotonus. The normal response of nerve and muscle to electricity has already been discussed (see page 21) and will not be described again. In order to study the electrical reactions in the clinic a definite but simple technique is essential. First, the poles or electrodes should be widely separated so that they do not interfere with each other. The electrode which is to be used to excite the muscle is called the *active* electrode; it is held preferably in a handle which contains an interrupter so that the current

can be turned on or cut off instantly at the operator's will. The other electrode must be kept entirely away from the field of observation, and hence is called the *indifferent* electrode. It should consist preferably of a large flat sponge, fastened upon a metal plate about six inches square; it can be held firmly by an attendant upon the patient's sternum. Both electrodes should be of metal covered with sponge or absorbent cotton, and this latter substance as well as the skin should be thoroughly wet. When using faradism it is of course not necessary to observe the polarity, but when using galvanism the poles should be changeable by an alternator on the keyboard; thus the observer has complete control of the current and the poles all in one electrode. To increase current strength, most keyboards now have appropriate devices; care must be used, however, not to give painful shocks. A water rheostat can be used to obviate such shocks when increasing the strength while using strong currents.

The first change noted in degenerating muscles is a gradual but rapid decline in faradic irritability. This begins usually in a day or two, and in marked cases the extinction may be complete in a week, or even less. Bowlby,[*] from accurate study of a case of nerve section in man, found that the faradic excitability of the nerve trunk disappeared entirely by the third or fourth day; whereas Erb states that it gradually disappears in the third week in animals. Such complete extinction denotes that the nerve influence is cut off absolutely. The reason for this, as explained elsewhere, is that the muscle normally has no faradic irritability of its own, and only responds to this current by virtue of the faradic irritability of the nerves within its tissue. Consequently when, in case of a nerve lesion, faradic irritability is abolished in the muscle, the conclusion is safe that the nervous influence is entirely cut off. This abolition of the faradic irritability is one of the early symptoms of neuritis and of pressure; it is seen especially well in Bell's palsy of the facial nerve, and in pressure palsy of the radial. In order to observe it accurately, the reaction of the muscle should be compared with that of its fellow on the opposite side; if both are paralyzed, the comparison should be made with some other unaffected muscle. Of course in cases in which the abolition is complete comparisons are not necessary. Either a slow or a rapid interrupter may be used; in small muscles a rapid one is rather the better, and in weakened muscles, in which the reaction is as yet only partly abolished, it is by far the better.

The reaction of the degenerating muscle to galvanism is much more complex than to faradism. Taking as a type a case in which the nerve influence is rapidly and completely cut off, as by section, the phenomena are as follows: The irritability of the muscle

is at first slightly decreased, then increased—*i.e.*, the muscle will respond to a weaker current than normally. This irritability diminishes again, and in a few weeks falls below the normal until in time it entirely disappears, just as did the faradic, but in much longer time. These changes are called *quantitative* changes. As the irritability decreases, the reaction of the muscle to the two poles changes. As already explained (page 26) the normal formula is:

$$CCC > ACC = AOC > COC.$$

This formula means that the cathode at the closure of the current is the most stimulating; next, the anode at the closure of the current; next, the anode at the opening of the current; and finally the cathode at the opening of the current. This normal formula is subject to only one variation in health, viz., the AOC is sometimes greater than the ACC, especially if the electrode be applied directly to the motor nerve. In the great majority of cases, however, the normal formula is as given. The degenerating muscle presents certain changes in this formula. First, the cathodal closure contraction (CCC) diminishes in force and as it does so the anodal closure contraction (ACC) increases or at least does not diminish proportionately, so that the two may equal each other or the anodal closure contraction may be even stronger than the cathodal. The formula is then written as follows for what may be called the first stage of degeneration:

$$CCC = ACC > AOC > COC.$$

For a more advanced stage, when the anodal closure has become the most stimulating, the formula is as follows:

$$ACC > CCC > AOC > COC.$$

Finally, according to some observers, the cathodal opening contraction (COC), which is practically never seen in health except with exceedingly strong currents, may appear unusually active in the degenerating muscle, and may even surpass the anodal opening contraction (AOC), so that the formula for a still more advanced stage is as follows:

$$ACC > CCC > COC > AOC.$$

With reference to this last formula I can only say that in a large number of observations, extending over years, I have rarely seen it. In fact I have but seldom seen the cathodal opening contraction (COC) as a result of disease.

As the muscle continues to degenerate, this reversed formula does not change again, but the weaker reactions gradually disappear, the ACC being the last faint response of the expiring muscle.

These serial changes are called the *qualitative* changes. They have been investigated especially by Erb [46] and called by him the *reac-*

tions of degeneration (RD). They are subject to some modifications, which are called partial RD and which should be carefully considered.

A third change noted in degenerating muscle is the *modal* change. This is highly characteristic, and as it may be the only notable change in some cases it has great importance. It consists in an alteration in the manner or mode of the response of the muscle to the galvanic current. In normal muscle still innervated by normal neurons the response is quick and complete and subsides at once. The more quickly the current is interrupted or alternated, however, the more active the response of the muscle becomes. In degenerating muscle cut off from its neurons all this is changed. The muscle no longer responds promptly and in its full length to the current; sometimes in fact only that portion of it lying immediately under the electrode responds, and then in a sluggish way that is in striking contrast with the normal activity. Moreover, the degenerating muscle does not respond readily to rapidly and oft-repeated breaks in the current; on the contrary, it rather requires a prolonged application of the electrode so that the current may run for some appreciable time—in other words, it responds better to slowly interrupted currents. A final characteristic of this modal change is the tendency of the sluggish contraction to endure the whole time the current is running between the interruptions. This is a sort of tetany, the cause of which it is difficult to explain. As it happens during the passage of the current it is called the "duration tetany," and its symbol is DT.

The "reactions of degeneration," as just described, are muscular phenomena entirely. It must be recalled that in health the reactions to both faradism and galvanism are due to the excitability of the neurons, *i.e.*, the axis cylinders in the nerve trunks, but that in the degenerative cases which we are now considering these nerve trunks are no longer in a normal state. When the neurons are degenerated they lose their excitability to all stimuli; hence in degeneration the nerves early lose both their faradic and galvanic irritability.

All these changes in the reactions of degeneration are graphically shown in the accompanying diagrams from De Watteville.

In Fig. 13 motility is lost at the first line (marked *o*). The galvanic excitability falls slightly in the first week, then rises, and finally sinks to normal; the wavy part of the line indicates the qualitative changes already described. The faradic contractility of the muscle falls and is soon entirely lost. In the nerve the excitability to both faradism and galvanism indicated by a joint line sinks also rapidly and disappears. Motion returns in the paralyzed muscle at the point marked by the star on the line marked *b*. At this time the nerve is regaining its excitability to both currents, and the muscle conse-

quently is beginning to react to faradism. The qualitative changes (RD) continue, however, for some weeks longer, in accord with an interesting and almost invariable rule that the degeneration in the muscle does not entirely disappear for some time after its nervous connections are restored. This diagram is of a case of complete but not long-continued degeneration.

In Fig. 14 the reactions of degeneration are shown in an incurable case of nerve injury. Motility is lost at the line marked *o*. The muscle shows a slight initial decrease in galvanic excitability, followed soon by the characteristic increase and the appearance indicated by the wavy line, of the qualitative or serial changes. This galvano-excitability then gradually declines until it becomes extinct after many weeks. The farado-excitability of the muscle falls quickly and disappears in three weeks. In the nerve the electro-excitability for

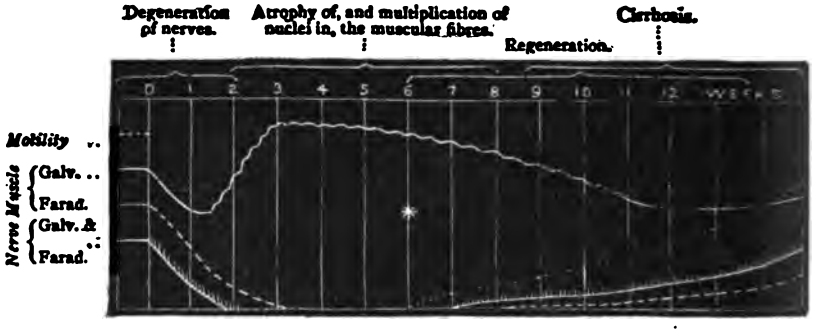

FIG. 13.—Diagram Showing Changes in RD in a Case of Trophic Lesion with Recovery. (De Watteville.)

both currents falls even more quickly, and is lost in the second week. No recovery from the paralysis occurs; the muscle permanently degenerates, and the electro-excitability in both nerve and muscle is ultimately lost forever.

These diagrams, as De Watteville says, are intended to represent typical cases merely. These are cases in which the nerve influence is cut off entirely, as, for instance, by a complete section of the trunk of the nerve, or by a completely obstructive lesion, as, for instance, a localized inflammation, such as occurs frequently in the seventh nerve. In all such cases the phenomena for the first few weeks are identical, and only vary later according as the injury remains permanent, as in section, or a cure results, as in inflammation. These variations are shown in the diagrams, one of which is from a curable, the other from an incurable case.

Many cases, however, are not typical, and these present variations from the classical reactions of degeneration. Such cases are those

especially in which the degenerative process is not complete; hence there is displayed a partial RD. It may be supposed that in numerous cases, both of nerve injury and of inflammation, the lesion is not completely destructive. There may be sufficient injury or pressure to interfere with free and full passage of nerve impulses, but not sufficient to shut them off completely; or some axis cylinders may be more injured than others. In such instances—and they are numerous in the clinic—the muscle or muscles are not completely paralyzed, and recovery may not be long delayed; and it is in these cases that the classical reactions of degeneration often fail to appear.

This partial reaction of degeneration has a number of varieties. In the mildest cases there may be merely a slight diminution in faradocontractility, noticeable on comparison with the corresponding muscle on the opposite side. In more severe but still mild forms the faradocontractility may be abolished and the galvano-excitability of the

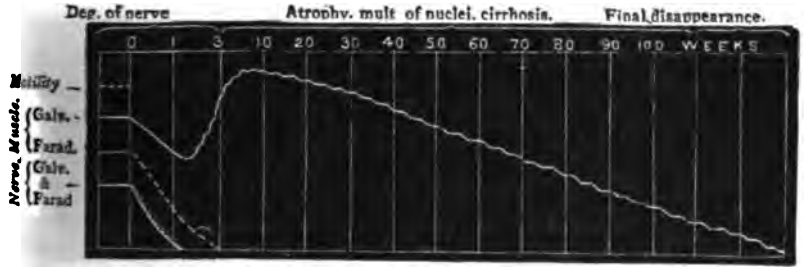

Fig. 14.—Diagram Showing RD in a Case of Trophic Lesion Without Recovery. (De Watteville.)

muscle modified merely in the modal change already described. Thus the muscle gives a sluggish response and shows a tendency to the duration tetany (DT), which is highly significant. This may not be accompanied with true serial or qualitative changes and yet it is an invaluable indication of some degree of true degeneration. Erb [80] describes a partial RD in which the muscle displays both the quantitative and qualitative changes, but in which the nerve does not entirely lose its electro-excitability to either current. In such a case the injury to the nerve is evidently but partial, and while preventing the passage of the impulses upon which depend the motor and trophic functions, is not sufficient to oppose the passage of impulses excited by the active stimulation of electricity. Such cases are sometimes seen in Bell's palsy and in various pressure palsies.

It is not worth while to point out all these minute and manifold departures from the type. They can be recognized clearly and their true value estimated only by one who understands thoroughly the typical reactions of degeneration already described.

The significance of the RD in the clinic is very great. These reactions always show infallibly that the muscular tissue is degenerating because it is deprived of the subtle trophic influence of the neuron. This may be caused, as already explained, by any lesion whatsoever that cuts off the muscle from that part of the neuron containing the nucleus, *i.e.*, the large multipolar cells in the anterior cornua of the spinal cord. Hence the RD is always an indication of an affection of the cerebrospinal nerves or of the anterior horns of the spinal cord, or, to state it concisely, of the peripheral motor neurons. These reactions never occur in cases of cerebral palsy from any cause, as hemiplegia from capsular hemorrhage; or in cases of diseases of the spinal cord that do not involve the anterior horns, as descending lateral sclerosis, locomotor ataxia, or transverse myelitis above the seat of the neurons. Whatever apparent exceptions to this rule occur in such diseases are satisfactory proofs that for some reason the disease process has spread so as to include these peripheral motor neurons. On the other hand the RD, partial or complete, is a usual accompaniment of acute anterior poliomyelitis, of all forms of neuritis, and of all kinds of injuries to nerves.

While, therefore, the clinical significance of these reactions is fully apparent, it does not necessarily follow that their exact pathogeny is very clearly understood. In other words, there does not appear as yet any sufficient explanation why degenerating muscular tissue should present these changes in its electro-excitability. It cannot even be said that it is known exactly what stages in degeneration, *i.e.*, what minute histological changes, underlie the various stages of the RD. From what is known of degeneration of the muscular tissue, it can only be surmised that the changes in the protoplasm of the muscular fibre determine in some way the altered electro-excitability. But to explain this seems to be as hopeless as to explain any other of the occult qualities of organic matter. The clinical facts already given, however, are practically all that concern us in this place.

The diagnostic importance of the RD is now apparent from what has been said. The RD is of absolute importance in determining within certain limits the seat of the lesion, *i.e.*, it indicates always an affection of the peripheral motor neuron. It is only of relative importance in determining the nature of the process taking place in the neuron, *i.e.*, it merely indicates that this process must be either destructive or obstructive. It is finally of no practical importance in determining the cause of the lesion, which, therefore, must be sought for in other clinical signs and in the history of the case.

In estimating the electro-excitability of nerves and muscles a knowledge of certain normal variations is essential.

First, it is important to know that all the superficial nerves of the human body are not excitable in health to exactly the same extent. These variations probably depend largely upon the degree of resistance offered by the surrounding and overlying tissue of the nerve-trunks. This factor, however, may not be the only one, for the individual neurons in some nerves may, for aught that is known to the contrary, be more excitable than those in other nerve trunks. The clinical fact is as stated. The most excitable of the superficial nerves is the spinal accessory. This fact can be tested by applying a weak faradic current to the various nerves of the body in turn, and gradually increasing its strength; such a current, slowly increased, will excite a response in the spinal accessory nerve before it does so in any other nerve. This nerve lies very close to the surface, and the skin overlying it is softer and more delicate than that overlying some other nerves; these facts evidently have an important bearing on the subject. In making such tests, however, it is absolutely essential that the skin and the electrodes should be equally wet, that pressure should be as evenly gauged to each nerve as possible, and that all the conditions should be as nearly the same as possible. The degree of excitability of the several superficial nerves has been variously estimated, and may not be absolutely the same for all individuals. Conditions as to fat, thickness of skin, etc., vary so much among persons that no table can be absolutely fixed. The following, however, from Hughes Bennett[81] is believed to be as nearly accurate as can be attained:

Spinal accessory.
Branch to levator anguli scapulæ.
Ulnar.
{ Median.
Facial.
Cervical plexus.
Anterior crural.
External popliteal.
{ Musculo-spiral.
Internal popliteal.

As shown in this table some nerve trunks have practically the same degree of excitability; for instance, the median, facial, and others included in the bracket. This fact is important for purposes of comparison. For example, as Bennett points out, if both lower limbs are paralyzed it is impossible to compare the degree of irritability of one external popliteal with that of the other, but as in health this irritability is equal to that of the facial and median, these latter nerves may be used as standards for comparison.

Second, the *motor points* of the muscle are its most excitable parts. A motor point simply marks the place of entrance into the muscle of its motor nerve. These points are not quite so excitable as the nerve trunk itself, but they are more excitable in health than the muscles themselves. In fact, being the points of entrance of the nerves into the muscles they display nerve irritability rather than muscle irritability; consequently in degeneration, when the neurons have ceased to respond to any form of electricity, these points are not more excitable than other portions of the muscles. In health they may be said to occupy an intermediate place, with reference to excitability, to the nerve trunk and the muscle proper. The farther they are located from the nerve centres the less excitable they become. For comparison the ulnar nerve may be tested; the nerve trunk at the inner condyle of the humerus will be found rather more excitable than the motor point of the flexor carpi ulnaris.

The muscles themselves, outside the influence of the motor points, are much less excitable than at these points, but they vary very much among themselves and among individuals. These variations probably depend upon the richness of the nerve supply to the muscle, *i.e.*, the number of nerves ramifying in the substance of the muscle, the richness of their arborizations, and their proximity to the surface. No definite rule and no systematized table can be given for all these variations in the human body. As a general rule it may be said that the larger muscles with coarser fibres (as the gluteus maximus) respond less readily than do the smaller muscles of finer texture. In health it must be remembered the response of the muscle, even when the electrode is applied directly to its body and not to its nerve or motor point, represents the irritability of the nerves within its structure; hence the response in health differs in *mode*, *quality*, and *quantity* from the response in degeneration, when the nerve supply is cut off, as has been already explained.

Trophic Lesions of the Skin and its Appendages.

The disorders of nutrition in the skin, nails, and hair caused by diseases of the peripheral nerves are numerous and important. The recognition of these trophic changes is still comparatively recent. In 1831 Bright (quoted by Wood") noted the possible relationship of herpes zoster to disease of the nerves. This seems to have been merely a shrewd speculation rather than a scientific demonstration, but it was one of the earliest hints in literature of a great truth. Before 1849 Brown-Séquard following S. van der Kolk, had experimented by cutting the sciatic nerve in the rabbit. In 1857 Parrot claimed that herpes or zona was a secondary manifestation of certain

neuralgias. Charcot[9] in 1859 was the first who demonstrated the connection of zona with a wound of a nerve. Later this observer and others (Bärensprung *et al.*) demonstrated the pathological anatomy of the nerve trunks and spinal ganglia upon which the symptoms depend. It thus appears that herpes zoster was the skin lesion that first drew attention to the trophic influence of the nerves upon the integument, and that this recognition is closely associated with the name of Charcot. Even at that date the peripheral sensory neuron which has its seat in the intervertebral ganglion was clearly but unwittingly shown to be implicated in the disease process. Bowlby[10] states that Hilton, in his lectures on "Rest and Pain," was, with Paget, the first author to draw attention to the altered nutrition of the parts supplied by a divided nerve; but this reference by Hilton can scarcely claim precedence over Charcot's case.

The further and complete elucidation of this subject owes much to the Civil War in America. This war gave unequalled opportunity for the study of nerve lesions, which strangely had been almost ignored in former great conflicts. To the genius and industry of Mitchell, Morehouse, and Keen, who had the insight to seize this great opportunity, science owes the first attempt at a complete presentation of this subject on a large scale.

Among other early observers Paget,[11] who demonstrated the "glossy skin" of neuritis, and Hutchinson,[12] who observed the pemphigoid eruption which follows nerve injury, deserve special mention. After the American Civil War, which had introduced this subject to the scientific world on such an heroic scale, the literature of trophic disorders increased rapidly, and now forms a library in itself. Reference to the more important and original of these works will be made under separate headings. (See also Vol. V. of this series.)

Brown-Séquard was the first to insist upon a distinction between the effects of destructive and those of irritative lesions. Some of his distinctions are erroneous, as for instance in the case of the muscles; but others, especially in the case of the skin, have some reason for them. Mitchell inclines to the view apparently that there is an element of "irritation" necessary to produce trophic lesions in the skin. He says that these lesions seldom appear early in cases of nerve injury, and that they are most likely to occur in cases in which the wounds fall into inflammation. As they do not arise in all cases of nerve injury he seems to imply that such injuries require some form of irritation to cause cutaneous symptoms. He furthermore says that trophic lesions never follow nerve injury unless there is also some motor or sensory paralysis; in other words, the trophic function is never the only one to be affected. This author, indeed,

says that trophic lesions in the skin are always accompanied with some disorder of sensation, either dysæsthesia, anæsthesia, or hyperæsthesia. As we have seen already that there is no evidence whatever of distinct trophic neurons, this clinical observation of Mitchell is in accord with the most reasonable theory, namely, that the ordinary neurons of motion and sensation have trophic functions. It is an undoubted fact that many observations of nerve injuries had been made and accurately recorded before the American Civil War. The names of Earle, Bell, Cline, Hamilton, and Astley Cooper in Great Britain, and of Feron in France, are associated with such cases (Avezou[46]). This fact, however, does not detract from the originality and value of the work by the American army surgeons.

Recently the tendency among some physiologists has even been to deny the trophic influence of nerves. Thus Krause, from the results of excision of the Gasserian ganglion and of the trunk of the fifth nerve centrad to it, concludes that there is no such function. Turner, after experiments, came to apparently similar conclusions.

The trophic disorders of the skin and its appendages (the nails and hair), and of its underlying connective tissue, may be divided into groups as they present themselves in the clinic. Only those that are associated with diseases of the peripheral nerves will be described. A somewhat large group of trophic disorders, both in the skin and in the joints, is as yet almost exclusively associated with diseases of the nerve centres. These also probably depend in fact upon disease of the peripheral neurons, and so naturally should be included in the groups made up of those which we are considering. Thus acute decubitus and arthropathy evidently depend upon affections, in part at least, of the peripheral neurons either in the spinal cord or the intervertebral ganglia, yet so far as we know at present they are not seen in diseases of the peripheral nerves exclusively. To attempt to give a reason for this difference would lead to mere speculation, but it would seem to depend upon the fact that for the production of such trophic lesions as spinal arthropathy the involvement of the spinal connections of the neurons in the central gray matter is essential.

Pathogeny.—Before proceeding to a description it is worth while to consider the possible action of two alleged contributory causes for trophic lesions, viz., disuse and injury.

Disuse cannot be conceived of as a cause of such skin lesions as large vesicles, like pemphigus and bullæ. However plausible the theory of disuse may appear in case of the muscles (and there it is easily disproved by the phenomena of degeneration), it certainly receives little if any countenance in case of lesions of the skin. The

œdema and glossy skin might be supposed to be due to a sluggish circulation due to disuse—but if this were so, why are not these phenomena seen in cases of paralysis from lesions of the brain and cord? The desquamation not infrequently seen in long-standing cases may indeed be in part secondary to disuse, to altered secretion of sweat, and to difficulty or neglect in taking care of the limb; but with this possible exception little if any importance can be attributed to disuse exclusively.

Injury as a contributory cause of trophic lesions cannot be lightly ignored. Patients with anæsthetic limbs may readily receive hurts of which they know nothing. A patient with syringomyelia in a Philadelphia clinic was known to have burnt his fingers without his knowledge because of his thermoanæsthesia. In such a case the injury, acting upon a limb in which the trophic functions are impaired, might readily act as an exciting cause. But these lesions, once started, often proceed to frightful mutilations, as the loss of part of a finger, which would not be the case probably in a healthy person. Bowlby noted in some of his patients that injury was the starting-point of an ulcer or inflammation of the finger. A blister recklessly and improperly applied to the region of the spine in some cases of acute disease of the spinal cord has been known to result in an ugly and obstinate sore. In all such cases, as is readily seen, what ordinarily would be a slight injury results in a serious one. The conclusion is warranted that trophic lesions may have their starting-point in external injury, but such injury is neither essential nor common in these cases. A strictly speculative paper on the theories of trophic lesions has been published by Poore.[33]

The most common trophic disorders of the skin are erythema, glossy skin, herpes zoster, urticaria, pemphigoid and bullous eruptions, eczema, œdema, phlegmon, destructive whitlow, and malformations and shedding of the nails.

Erythema and Glossy Skin.—One of the commonest as it was one of the earliest of these lesions to be observed is erythema with glossy skin. These are so intimately associated with each other and with œdema that it is difficult to describe them apart. Paget[34] was among the first to call direct attention to this condition; since he wrote it has frequently been recognized. Before Paget, however, Hamilton[35] had referred to the redness and œdema of the skin in a description that was lifelike. The onset and appearance are somewhat as follows: After nerve injury or disease symptoms of irritation are present, such as neuralgia. This neuralgia has often a characteristic quality; it is a burning pain as well as an ache, hence it has been called *causalgia* (from the Greek words καῦσος, a burning sensation, and ἄλγος,

pain). The limb, or the part especially supplied by the affected nerve, as for instance the fingers, becomes swollen and mottled, or, as Paget wrote, "blotched as if with permanent chilblains." The œdema is often quite tense and brawny. The skin becomes smooth, glossy, devoid of wrinkles and hair, and usually unnaturally dry. In some cases there is a tendency to shed the epithelium. The fingers become tapering and the nails may begin to show trophic changes. The parts, as already said, are the seat of severe pain, and must be touched and handled with care. Mitchell insisted that this state of the skin was never present without burning pain, a statement to which Bowlby takes exception. But certainly even if exceptions occur the rule usually holds good.

Glossy skin may appear early and may continue for a long time. Bowlby saw it in a case in which it had lasted for twelve years; in another case it had lasted ten years. In some cases in which pain is no longer a prominent symptom causalgia may recur at intervals, especially in cold weather. Paget says that glossy skin may follow "shingles" or ordinary herpes zoster, as well as common neuralgia. In the latter case this would be a clear sign that the nerve was the seat of some definite irritation. When long continued, glossy skin is rather an unfavorable sign, as it indicates of course an intractable state of the nerve; but in many cases it is not long continued, and, on the whole, as Mitchell says, the prognosis is favorable.

Œdema.—This is not an unusual result of inflammation and injury of nerves. In multiple neuritis œdema of the legs is very common. In injury of single nerve trunks this condition, while not so common, is still common enough to be a sign of importance. In polyneuritis it appears as a rule rather promptly, at least it is usually well seen by the time the patient is ill enough to take to bed. In case a single nerve is injured, the swelling may be limited to the area of its distribution, and if this swelling is accompanied with pain and alteration in the color of the skin the resemblance to an acute inflammation or phlegmon may be misleading. In Hamilton's early case, already quoted, the back and palm of the hand were swollen and painful and the skin was reddened, so that the appearance of commencing abscess was simulated; but this swelling and redness disappeared over night, to be repeated subsequently. Watson, according to Mougeot, cut deeply into such a swelling, mistaking it for an abscess. In some forms of neuralgia, especially of the trigeminal nerve, swelling of the subcutaneous tissue is seen.

The cause of this œdema is not absolutely known, but it probably depends, in part at least, upon paralysis of the vasomotor nerves.

Urticaria.—This is not a common trophic lesion. Charcot men-

tions having seen an eruption of urticaria follow the fulgurant pains of tabes. I have never seen this, and I do not recall any other reference to it.

Herpes.—An herpetiform eruption, as already said, was the first variety of trophic lesions of the skin to attract attention. It is characterized by a group of vesicles on an inflamed base. It is usually accompanied with symptoms of irritation, such as intense burning pain, neuralgia, etc., just as in the case of true herpes zoster. Some observers, as Charcot, who cling to the distinction made by Brown-Séquard between destructive and irritative lesions, say that herpes is seen only in the latter cases; that this eruption never follows clean and complete section of the nerve. Charcot's case, which was the first on record, occurred in a man who had received a bullet wound in the thigh. It has been debated whether herpes ever follows a purely central lesion of the nervous system. Cases have been reported which seem to show that it can follow such lesions. Schmidt, quoted by Ross," had a case of injury to the lumbar spine in which an herpetiform eruption came out along the courses of the first and second lumbar nerve; but this may have been, and probably was, due to injury and irritation of the nerves and nerve roots within the spinal canal or at their exits. Herpetiform eruptions undoubtedly occur in locomotor ataxia, but the peripheral sensory neurons are so much involved in that disease that the trophic lesion may well be supposed to be caused by an irritation of them; moreover, in tabes this eruption usually accompanies or follows crises of fulgurant pains and appears along the course of those nerves which evidently have been the seat of these pains. A very few cases of hemiplegia have been reported in which herpes appeared promptly on the paralyzed side, but these cases are open to the grave objection that apparently no post-mortem examination of the nerve was made. In one case of hemiplegia followed by herpes, reported by Charcot, it was found that the hemiplegia was not the cause of the herpes but that the eruption evidently depended upon irritation of the ganglion on the posterior root of one of the sacral nerves, due to an embolus in a contiguous artery. The evidence is in favor of the theory that herpetiform eruptions are due to some irritative lesion of the peripheral neurons; and, as the symptoms of irritation are usually marked by pain, etc., that the sensory neurons especially are implicated.

This "traumatic" herpes or zona appears usually rather late, although this is not an absolute rule. Depending, as the lesion probably does, upon irritation of the nerve, it occurs only after such irritation is set up, and the time for this of course varies. It may occur after the original injury has healed, or other exciting cause has ceased

to be operative. According to Mougeot[40] neuralgic symptoms are not essential in symptomatic herpes, for a few cases have been observed in which they were absent. In many cases the eruption is recurrent. It usually follows the course of the nerve or appears in the territory supplied by the ramifications of the nerve, and observations to the contrary are open to doubt.

The question arises, Is traumatic or symptomatic herpes the same thing as the herpes zoster or zona of the dermatologists? There seems to be no doubt that the two are practically identical so far as their clinical aspects go. The two forms differ apparently only in the fact that one is due usually to trauma, the other to an unknown source of infection; the essential cause, irritation of either the nerve trunk or ganglion, is the same in both. In ordinary herpes zoster the mode of introduction and exact manner of action of the cause are obscure; this may be even microbic in nature, for aught that is known to the contrary. In the traumatic variety the introduction of the cause is not so obscure, although its exact nature, whether microbic or not, is as much unknown as in the former instance. Bärensprung[41] found the spinal ganglia affected and the intercostal nerves thickened and injected in herpes zoster; and a similar pathology seems to be indicated for the traumatic or symptomatic variety.

The spinal ganglia, according to Edinger,[42] have been found diseased only in a few cases, but according to our observations these cases are now sufficiently numerous to establish the important fact of a direct etiological relation. The interesting cases, for instance, of Wiss and Wiedener, in which herpes zoster had affected the eyeball, and autopsies had revealed extensive diseases of the Gasserian ganglion (referred to under the heading of diseases of the fifth nerve), are cases in point. According to Edinger the physiological observations of Gaule indicate that certain elements of the spinal ganglia have a vasomotor trophic influence on the skin and muscles. He thinks that these may be sympathetic fibres which in great numbers surround the cells. According to Vulpian, Wollenberg, and others, important changes in the spinal ganglia have been observed in tabes not associated with herpes or similar lesions.

Pemphigus.—In addition to herpes there may be other vesicular eruptions, the most common of which is pemphigus, or pemphigoid bullæ. Hutchinson, Charcot, Weir Mitchell, and other early observers noted this. These bullæ may appear either early or late in the case. Mitchell saw the thumb covered with a large bulla on the fourth day after injury to the nerve at the wrist. Bowlby says that vesicular eruptions are common for some months after the primary lesion, but that he had not seen or heard of such symptoms appear-

ing in old-standing cases. But I have recently reported a case of spina bifida in which trophic or symptomatic pemphigus was seen. The case is as follows[*]:

C. E——, female, aged 9 years, had a spina bifida of the lumbar region, involving the first three vertebral arches. She was paralyzed in her legs and had badly deformed feet, presenting an extreme varus. There was some wasting of the muscles below the knees. There was slight foot clonus on the right, none on the left. The patellar reflexes were abolished. The patient had no control of the bladder, and diminished control of the rectum. The tactile sense was very blunt below the knees, if not abolished. Above the knees a line of demarcation of the anæsthesia was well marked; slight sensation was preserved on the inner side of the leg. The thighs were sensitive. To heat and cold sensibility below the knees was abolished, above the knees it was normal. All the muscles of the thighs and legs responded normally to the faradic current, except the peroneal group. To galvanism in strong currents there was complete abolition of response in the peroneal group. Slight modal change was noted in the flexors of the big toe. The mother reported that the child was liable to the formation of "bladders" on the legs, *i.e.* bullæ, leading to trophic sores. These sores left scars after healing. In the bullous stage they resembled pemphigus. On the girl's admission to the Home for Crippled Children one of these trophic suppurating sores was seen on the left great toe, looking almost like a perforating ulcer. The fronts of the tibiæ were marked by the scars of these sores.

In considering this case, the following points are important. The type of spina bifida was meningomyelocele, in which type the involved portion of the spinal cord is not properly developed and the nerve trunks are implicated in the diseased mass which protrudes from the opening in the spinal canal. Some, but not all, of the neurons were involved in this case, as shown by the anæsthesia, altered electrotonus, paralysis, atrophy, etc. The case was congenital, hence of long standing. The trophic lesions occurred without signs of irritation. The bladder and rectum were involved, evidently from deformity in the cord itself.

As in this case, pemphigus, when the bulla breaks, discloses an ulcerating sore, which may heal up in a short time, or may be more intractable and even lead to quite extensive necrosis of tissue. In either case it leaves scars. Hutchinson noted that extensive whitlow may result from such a bulla. Pemphigoid eruption, like herpes, is more apt to result from wounding and irritation of a nerve than from the complete section of it. Ross cites a few instances in which bullæ appeared in cases of disease of the spinal cord, but in none of them is it plain that this lesion was not due to irritation of the nerve roots. The case from Chvostek, in which pemphigus oc-

curred in the fourth week after a hemiplegia due to hemorrhage in the left temporal lobe and lenticular nucleus, is open to doubt unless the peripheral nervous system was carefully examined.

Eczema.—It is doubtful whether true eczematous eruptions occur as trophic sores after nerve lesions. A general consensus of opinion seems to range the vesicular trophic eruptions under the head of either herpes or pemphigus. These vesicular eruptions, especially the bullous kind, often constitute an early or initial stage of destructive lesions, such as ulcers, phlegmons, and whitlows. But these lesions are not properly eczematous in any of their stages. Some of the early observers described eczematous eruptions, but it is apparent from their writings that they referred to the phlyctenular and herpetiform lesions, often proceeding to ulcer and whitlows, already described.

Changes in the Nails.—Trophic changes in the nails are not uncommon. Weir Mitchell " was among the first to describe them. They are observed in central as well as in peripheral diseases. Thus they are seen in syringomyelia especially. Of peripheral affections they are seen most strikingly in cases of wounds of the nerves. In cerebral lesions, as in cases of hemiplegia, I have not noted the complete arrest of growth of the nails, of which Mitchell speaks; but the growth is probably somewhat retarded in these cases.

The various alterations in the nutrition of the nails, due to nerve lesions, are arrest or retardation of growth, curvature in the anteroposterior diameter, transverse and longitudinal fissures, grooves, and cracks, shedding of the nail, and hypertrophy and deformity.

The portion of the nail that is altered, as by grooves and furrows, is sometimes well demarcated from the healthy nail, the line of separation indicating the time or stage when the lesion occurred. Shedding of the nails is a common accompaniment or result of whitlows. It may occur, however, independently of whitlow, as I have seen it do in syringomyelia. S. van der Kolk cut the sciatic nerve in animals, and observed that, among other trophic changes, the nails were shed. The nail may also undergo an hypertrophy and be much deformed in shape. In some cases it becomes dry, brittle, and scaly. Trophic lesions of the nails may occur in cases in which there are no nerve lesions. I have under observation an artist, who for twenty years has had curious changes in his finger nails. These have become very thin and brittle, so that the edges cause great annoyance by catching on clothing and other objects, and thus tearing off down to the quick. Ridges or grooves have formed, not in the transverse, but in the longitudinal diameter of the nail, and a great central ridge divides the nail into two halves. By a singular coincidence this man has recently

developed a true scrivener's or draughtsman's palsy, due to overwork.

Changes in the Hair.—As already stated, falling of the hair is an accompaniment of glossy skin in that state which sometimes follows wounding of a nerve. In animals section of the sciatic nerve causes falling of the hair almost always, but this grows again even in cases in which the nerve is not regenerated (Arnozan[35]). The growth of hair may be increased in abundance by lesions of nerves. Thus Hamilton,[36] in 1838, reported a case of injury to the musculospiral nerve in venesection, in which the arm became covered with an excessive growth of hair. H. C. Wood refers to a case of arsenical paralysis, in which the legs became covered with a growth of hair. Such a growth is probably favored by disuse of clothing and exercise.

The abundance of the hair is not only affected in nerve lesions, but the character of the individual hairs may be altered. Thus the hairs may become thinner, or thicker, or more brittle than in health. In some instances of severe neuralgia the hair seems to become morbidly sensitive, but this is only apparent and is due to the hypersensitiveness of the underlying skin, for the hair itself is never sensitive.

Decoloration and alterations in color of the hair, as results of nerve lesions, are subjects of much obscurity. Cases of migraine and severe neuralgia, in which the hair has turned gray in limited areas, have been reported. While some of these cases appear too well supported to be denied, many of the sensational tales told of persons whose hair has turned white in a single night under the influence of emotion are probably spurious. H. C. Wood [37] has collected some of these tales, to which he seems to give credit. Byron, the poet, evidently believed in this *canities*, for he makes the prisoner of Chillon explain that his

> "Hair was gray, but not with years,
> Nor grew it white in a single night,
> As some has done from sudden fears."

Kaposi has denied, according to Arnozan, the possibility of such cases. The cases of alopecia, followed by a growth of parti-colored hair, are probably not due to nervous influence, as was formerly believed, but to parasites.

Trophic Lesions of the Bones.

Trophic changes in the bones, as results of nerve lesions, are very common, but the whole subject is still involved in much obscurity. These changes occur in cases as well of central as of peripheral dis-

ease. The several varieties of these changes, however, are not observed with equal frequency in both these classes of nerve lesions; in fact, some of the most important of these trophic lesions are seen to follow diseases of the spinal cord only. In consideration of the facts, however, that the peripheral nervous system is made up entirely of neurons which have their courses largely in the spinal cord, and that it must be by way of these neurons that trophic influences, for good or ill, pass, it is difficult from an anatomical standpoint to make the distinctions which, nevertheless, are patent in the clinic. Thus a so-called spinal arthropathy must be the result of an affection of the peripheral neurons; yet, so far as I know, this form of arthropathy does not show itself in cases of pure peripheral disease, as, for instance, neuritis. It thus seems that in order to produce this affection, the neurons must be involved in their course within the spinal cord.

Arnozan surveys this whole field, and classifies the following varieties of trophic lesions of the bones and joints. 1. In cases of arrested development, especially in cerebral atrophy or what we now call porencephalon, the bones and joints share in the failure to develop; contractures and a sort of fibrous ankylosis occur. 2. In the osteomalacia of the insane; this leads to fractures, so common in dements and chronic lunatics. 3. The bone lesions of locomotor ataxia — the true tabetic arthropathy; here also sometimes occur spontaneous fractures, with exuberant callus. 4. The bone changes seen in scleroderma, leprosy, and hemifacial atrophy. 5. The bone lesions following peripheral nerve injury. S. Weir Mitchell says that wounds of nerves can develop inflammatory conditions of joints, which "so precisely resemble rheumatic arthritis in their symptoms and results that no clinical skill can discriminate between the two." Such well-marked pseudorheumatic joints, following nerve injury, are not so common or so confusing in my observation as this statement seems to imply; but cases of very stiff and painful joints with fibrous ankylosis, following injuries to nerves, do undoubtedly occur. A not uncommon instance of this is seen in the shoulder-joint, following injury to the circumflex nerve, due to dislocation of the head of the humerus; in such cases the deltoid is paralyzed and atrophied, and exhibits the reactions of degeneration. 6. Finally, the articular lesions, pseudorheumatic, closely resembling those in the last group, which follow hemiplegia and some acute diseases and injuries of the spinal cord. Gull,[4] in 1856, reported the case of a man, aged thirty years, who had a tumor of the upper dorsal cord, and whose symptoms simulated phthisis pulmonalis. Gradually increasing paraplegia with incontinence came on. He had painful joints, and these led to

the erroneous diagnosis of rheumatism. In a second series of cases, Gull reported two other cases of cord lesions with rheumatoid joints. This reminds us of the fact that, in 1831, John K. Mitchell[84] advanced the theory of the spinal or nervous origin of rheumatism. Talamon[85] makes the following distinction: in locomotor ataxia the arthropathy begins in the bone, the nature of the disorder being still unknown; while in hemiplegia, cord lesions, and nerve wound there occurs an arthritis beginning in the synovial membrane. I am not sure that this distinction is founded on fact, but, so far as nerve lesions are concerned, the arthropathy, as Talamon says, appears to be limited in the main to the membrane.

From the above it appears that of the variety of bone lesions following nerve lesions, a small proportion are due directly to disease or injury of the peripheral nerves. As we are concerned here entirely with this last-mentioned subject, I shall describe these particular bone lesions only.

Experiments on the lower animals have tended to prove that injury to the nerves causes changes in the bones. Schiff (quoted by Ogle[86]) found that after dividing the crural and sciatic nerves of a dog, "the bones of the paralyzed side are less voluminous than those of the opposite side, while the periosteum of the paralyzed side is thickened and composed of many layers, often numerous and easy of separation." Moreover, "the diminution of the inorganic component parts had so far proceeded that the neck and lower end of the femur and the upper end of the tibia had become quite cartilaginous, soft, and flexible." Later the bones hypertrophied, especially in young animals. Schiff concluded that the immobility of the muscles caused the atrophy of the bone, the hypertrophy being caused by alteration of nutrition arising from paralysis of vascular nerves. Ollier found that the incisor teeth in a rabbit fell out after division of the maxillary nerve. He also observed that section of all the nerves in a limb caused shortening of the bones.

In man clinical observation proves undoubtedly that injury of the nerves causes changes, especially in the joints. Weir Mitchell states that these arthritic changes may appear early or late. A large articulation may be involved, or all the small joints of the fingers. The swelling and redness are usually slight, but the joints are exquisitely tender on touch or motion. This state may remain for weeks or months; when it declines it leaves the joints stiff, somewhat enlarged, and painful on motion. Some of these cases recover, but in others a resulting ankylosis may occur, which is exceedingly obstinate to treatment. Bowlby says that slight changes in the joints are common. He thinks that the finger-joints are especially liable to be

affected, more so than the wrist and elbow, even in cases in which the nerve injury has been in the upper arm.

In brief, the changes commonly seen are stiffness, swelling, and slight redness, with ankylosis in the worst cases. The resulting condition is suggestive of chronic rheumatism.

The anatomical changes were studied by Bowlby in the case of a man who had injured his median nerve. The capsules of the finger-joints were thickened, and the cartilaginous surfaces of the most distal joint were united by fibrous tissue. The terminal joint of the ring finger was firmly ankylosed, the union being by bone. Microscopically, the normal hyaline matrix of the cartilages was replaced in part by fibrous tissue.

In a case of injury to the circumflex nerve, caused by a dislocation of the head of the humerus, I once observed paralysis and rapid atrophy of the deltoid muscle, with complete reactions of degeneration, and firm ankylosis of the shoulder-joint. This latter required to be forcibly broken up. The patient ultimately made a good recovery. In such a case, of course, the injury to the joint may have been partly due to the original dislocation, but it was probably promoted by the paralysis of the muscle and nerve.

The resulting trophic changes in the bone itself have been observed in man by Ogle, Blum, and others. Resulting atrophy is more common, apparently, in young persons. Ogle reports a case in which there was atrophy of the bones of the hand, and osseous union between the radius and ulna, following section of the median nerve. Complete destruction of the bones, *i.e.*, necrosis and discharge of the fragments, has already been mentioned as a result of the trophic whitlow seen in Morvan's type of syringomyelia, in anæsthetic leprosy, and in some cases of nerve injury. Valentin speaks of necrosis following an excision of a portion of the sciatic nerve for neuroma. Letiévant reported a case of necrosis of the extremity of the middle finger following section of the median nerve. In perforating ulcer of the foot (to be described), necrosis of contiguous bones is sometimes seen.

Atrophy of the bones of the leg, the femur especially, was seen by Lobstein in a man, aged fifty-four years, who had suffered in infancy from a grave wound of the thigh, involving both the sciatic and crural nerves. Those who wish to study this subject in detail may consult with profit the paper of Ogle,[47] and especially that of Talamon.[48]

The exact causes of these changes in bones and joints may still be the subject of some speculation. The resulting disuse of a joint tends, no doubt, to ankylosis, so that it is a question whether the whole of the lesion can be considered truly trophic. From a clinical stand-

point, however, these changes are closely allied to other trophic changes, so that the mere speculative question sinks into comparative insignificance. The important fact is that these arthritic lesions are not uncommon in cases of nerve injury. In cases of simple neuritis not due to injury—as, for instance, in multiple neuritis—stiffness and ankylosis are not uncommon when the disease is of a severe type and long standing. The joints become painful on pressure, but in my observation do not present quite such an irritative and rheumatoid aspect as they do in cases of mechanical injury to the nerves. True bone changes, such as atrophy, in cases of simple non-traumatic neuritis, are not observed; but destructive whitlow involving the bone may occur.

Trophic Lesions of the Subcutaneous Tissues.

Under this heading may be included whitlow or panaris, perforating ulcer, phlegmon and pseudophlegmon, and bedsore or acute decubitus. Some of these lesions are much more likely to occur in cases of peripheral disease than the others, and prominence here will be given to these.

Whitlow.—Destructive lesions, such as whitlow, perforating ulcer, etc., are sometimes caused by diseases and injuries of nerves. Schroeder van der Kolk, many years ago, cut the sciatic nerve in mammals, and noted that the limb became swollen, the toes ulcerated, and the nails were shed. Brown-Séquard repeated this experiment in guinea-pigs, and claimed that no change occurred when the limb was protected from rough contact with the ground. The attempt to trace these trophic changes to local injury has never been entirely successful. Hutchinson[35] reported seven cases of section of nerves, in five of which the fingers became inflamed soon after the accident. He argued against pressure or injury as a cause of the resulting whitlow, on the ground that this lesion occurred first at the tip of the finger, where there had been no pressure. He observed that the whitlow was peculiar, inasmuch as it thus involved the tip of the finger, both skin and subcutaneous tissue, and caused exfoliation of the nail. Paget, however, recognized the possibility of local injury acting as a cause, and reported ulcers of the fingers from burns received unconsciously in anæsthetic parts. This latter kind of sore, however, usually heals up as promptly in cases of nerve lesion as in normal tissue, and therefore cannot be called a true trophic lesion. On the other hand, the true whitlow, due to nerve injury, is usually a most destructive process.

As already explained, pemphigus is usually only a preliminary stage to an ulcerative process in the skin. When the bulla breaks it

reveals an ulcer, which in many cases may remain superficial, but in some cases may extend deeply. In either event it leaves a scar.

Whitlow, however, is much more than an ulcer in the skin. It is a deep-seated, destructive process in the subcutaneous tissue, usually beginning in the distal phalanx, and often involving and destroying the bone. It usually begins as an inflammation in the skin, in or near the finger tips; the subcuticular tissue is rapidly involved, and in some cases suppuration occurs quickly and may not be suspected by the patient, the pus being retained just beneath the skin. When this is evacuated the finger is seen to be extensively involved. Denudation of the bone occurs, and the phalanx may even be lost. In some cases more than one finger at a time is involved, and more of one finger than of another.

Trophic whitlow is almost always a painless affection, and this feature serves to distinguish it better than aught else. It seems thus, as in the case of so many trophic lesions, to depend especially upon an affection of the sensory neurons. Its time of occurrence, with reference to the original affection of the nerve, varies greatly. Cases are reported in which the bullæ have appeared in a few days after the nerve injury, and ulceration and gangrene have followed in a few weeks (d'Anson, quoted by Bowlby, whose reference I am unable to verify). In other cases the complication is delayed longer.

Injury to the artery as well as to the nerve is not an uncommon event, and some of the extensive sloughings reported by observers, as by Hilton" and by Bowlby, may possibly have been due in part to this accident. Still, this at best is but a contributory cause, and it is doubtful whether sloughing would occur in these cases if the nerve were not injured. For instance, sloughing of the hand as a result of ligation of the ulnar artery is not usually to be expected in cases in which the nerve supply is normal.

Among the diseases which especially exhibit destructive trophic whitlows are leprosy, syringomyelia, and the disease described by and named for Raynaud.

The form of leprosy which displays whitlows is the anæsthetic form. This variety, as is well known, depends upon a specific neuritis, which is caused by the bacillus of leprosy, discovered by Hansen. This disease is marked by initial pains, with areas of hyperæsthesia, followed by extensive anæsthesia. In time the nerve trunks become extraordinarily swollen, and the superficial ones may be felt as large and tender. Trophic lesions, as a rule, are early phenomena in these cases. They consist in macular and vesicular eruptions. The maculæ are highly characteristic. The vesicles appear as bullæ and pemphigus, such as have been already described, and proceed to

ulcerations, which may be terribly destructive. Both the fingers and toes are the seats of these whitlows, which not infrequently destroy the phalanges. Contractures and loss of power occur. The course of this form of leprosy is often extremely chronic. It evidently affords a striking example of trophic lesions depending upon affections of the nerves. The trophic process is practically identical with that which occurs after wounds of nerves; the only difference is in the essential cause of the nerve lesion. In the one case this is the irritation of the neurons, caused by a specific, virulent microbe; in the other it is the irritating effects of trauma.

Syringomyelia, a disease of the spinal cord, also causes trophic whitlows occasionally. The form of the disease that does this especially is the type originally described by Morvan, and sometimes called after him "Morvan's disease." Charcot,[10] in a lecture published shortly before his death, proposed to call this form the "type Morvan." He was among the last to concede that the disease, so well described by Morvan, was only a variety of syringomyelia. There can be little doubt now of the identity of the two affections. Joffroy[11] has shown by autopsy that a typical case, which had presented extensive mutilating whitlows, was one of genuine syringomyelia. The cases originally reported by Morvan[12] under the heading of "panaris analgésiques" presented the symptoms of paresis, with analgesia of the upper limbs, limited at first to one side, then passing to the other, and resulting in the production of one or many whitlows. Necrosis of the phalanges, especially the distal ones, was common. Morvan relates graphically how he discovered the analgesia in these cases. He had occasion to incise one of these whitlows, and was astonished to find that the patient, who did not seem to be a hero, bore the operation without a complaint. Investigation showed that the patient had suffered no pain. It is a singular fact that one physician like Morvan, in the province of Brittany, should have met with so many of these cases. In some cases there were initial pains. Other trophic lesions, as muscular atrophy, scoliosis, arthropathy, etc., were sometimes present. The special features of these whitlows are their grave but indolent character, and the facts that they are multiple and appear successively. Some cases are extremely chronic, lasting even as long as forty years. In some of these cases, as that of Gombault,[13] a peripheral neuritis has also been described; and this fact is important in relation to the etiology of these trophic felons. There can be no doubt that their general clinical appearances ally them closely to the whitlows produced by nerve lesions, and it is possible that even in a central disease like syringomyelia, which affects extensively the peripheral sensory neurons in

their course through the spinal cord, it may be by virtue of the involvement of these neurons that the destructive trophic lesions are produced. Certainly it is impossible to conceive of any other connection between these sores, located on the extreme periphery of the nervous system, and the disease process in the centre, except through a diseased peripheral neuron.

With reference to the exact morbid anatomy of Morvan's disease, while many of the observed facts indicate that it is a form of syringomyelia, it must be acknowledged that all the facts do not necessarily indicate the formation of *cavities* in the cord. There may in some of these cases be a process of gliomatosis in the central region of the cord that has not yet proceeded to cavity formation. This gliomatosis is the real disease—the cavity, of course, is only an effect of it. In such a case analgesia with trophic lesions, as well as other symptoms of syringomyelia (muscular atrophy, etc.), may exist, and the peripheral nerves, being but continuations of the neurons within the diseased portion of the cord, might be degenerated. This seems to have been distinctly the idea of Morvan himself, who says that the seat (*foyer*) of the disease is in that part of the cord which gives rise to the brachial nerves. Finally, it must not be forgotten that Morvan's disease, as well as other types of central gliomatosis, bears a striking resemblance sometimes to anæsthetic leprosy. The trophic lesions, especially the whitlows, are very similar in the two diseases. (For references to cases by Stendener, Langhans, Rosenbach, and others, see Bruhl.[35]) Some observers have even claimed the identity of syringomyelia with leprosy. This claim is perhaps somewhat strengthened by the curious fact that Morvan observed about twenty cases of his disease in a small fishing community. The identity could be established, of course, only by the discovery of Hansen's bacillus in syringomyelia and in Morvan's disease.

The curious disease described by Raynaud [36] has for its terminal stage symmetrical gangrene of the fingers and toes, and sometimes of the ears and nose. It has three stages: (1) local syncope; (2) local asphyxia; (3) symmetrical gangrene. The first stage is marked by a bloodless state of the parts, the second by a congested state, and the third by the gangrene. The affection is usually extremely painful. Hæmoglobinuria is sometimes present in the attack, and nervous symptoms, such as epileptic and hysterical phenomena, are seen. The cause and pathology of the disease are as yet quite unknown. It has been supposed to be due to disturbance of the vasomotor system. It is doubtful whether it is due to an affection of the sensory nerves, as instead of anæsthesia and analgesia there is usually pain. The gangrene, moreover, is usually symmetrical. Raynaud's disease

is mentioned here simply for the purposes of comparison and contrast with the trophic affections which are recognized as depending on lesions of nerves. It must be remembered, however, that peripheral neuritis has been observed in some cases; hence it may yet appear that the disease is only another nutritive disorder due to an affection of the nerves (Collins[40]). Krisowski ascribes a case of symmetrical gangrene in a child to syphilis. This suggests that the immediate cause is a specific endarteritis.

Perforating Ulcer.—Among trophic lesions this ulcer holds a prominent place. It was first described by Nélaton, in 1852, and was first called by its present name (*mal perforans plantaire*) by Vésignié. It consists, as its name implies, of a round or oval ulcer, perforating the skin and penetrating more or less deeply into the contiguous tissues. Ross suggests that it is really a sinus. It begins as a hard, corn-like elevation in the skin; this is in marked contrast with the bullous eruption which often ushers in whitlow. The induration soon breaks down in the centre and presents an ulcer. In mild cases this may heal rapidly; in severe cases it tends to perforate deeply, but not to extend in a lateral direction. In grave cases it may even involve the underlying bones and contiguous joints. This ulcer is usually associated with some degree of anæsthesia and analgesia. Duplay and Morat[41] found all the ulcers which they examined to be more or less insensible. I noted the same condition in the case about to be reported. Both modes of sensation usually are affected; in some cases, however, attempts at walking may cause pain. The extent of the anæsthesia will depend, of course, upon the extent and location of the nerve lesion. In cases of locomotor ataxia the appearance of the ulcer may be preceded by fulgurant pains.

Perforating ulcer may be accompanied with other trophic lesions, as shedding of the epidermis, alterations in the nails, œdema, phlegmon, and ankylosis and deformity of the neighboring joints. Its favorite seat is the under surface of the foot and toes; it is found especially on the ball of the great toe. It sometimes comes at the side of this toe.

Many theories have been suggested to account for this curious lesion. It has been ascribed to syphilis, uræmia, glycosuria, embolus, endarteritis, atheroma, inflammation of the sudoriparous glands, and other agents. There is now little doubt, however, that genuine cases of this ulcer are directly due to some nerve lesion. Dolbeau saw perforating ulcer of the foot in a case of fracture of the spine. Lucani saw one succeed compression of the sciatic nerve, caused by a fracture of the femur. Sézary saw one in progressive muscular atrophy. Fischer saw seven cases; one in myxoma of the spinal

cord, another in old hemiplegia, another following a wound of the sciatic nerve, another following luxation of the femur, another in vicious union of a fracture, another in gunshot wound of the buttock with injury of the sciatic nerve, and the last in a case of cord disease (tabes?). Mathieu also saw one follow a wound of the sciatic nerve. Locomotor ataxia, however, is undoubtedly the disease in which perforating ulcer in seen most commonly. Poncet thought that he saw a resemblance in the affection of the nerves in cases of perforating ulcer to that which occurs in anæsthetic leprosy. Estlander elaborated this idea. Duplay and Morat, in a careful study, concluded that perforating ulcer is associated with a degenerative lesion of the nerves. There thus seems to be no doubt that this ulcer is a true trophic lesion, depending upon an affection of the peripheral neurons, probably the sensory ones.

The following case occurred in my clinic at the Philadelphia Hospital.[52]

A. B——, aged 40, white. The patient had been tabetic for some years, presenting the usual sensorimotor symptoms. He presented a perforating ulcer on the inner and under surface of the great toe of the left foot. This was indolent, with little disposition either to spread or to improve. It was not painful; in fact, it was associated with considerable tactile anæsthesia and analgesia, as these ulcers, in my experience, are apt to be. It did not communicate with the bone or with the neighboring metatarsophalangeal joint.

It is possible that these ulcers are started in some instances by traumata, *i.e.*, by the bruises and pressure caused by an ataxic gait. The disease process, once started, is favored by the low vitality of the tissue, depending in turn upon the affection of the nerves. Hence, as has already been shown, it may appear both in central lesions of the spinal cord and in peripheral lesions, the peripheral neurons being equally involved in both instances.

Phlegmon and Pseudophlegmon.—Inflammation of the subcutaneous connective tissue, with formation of pus, is not a common result of nervous lesions. In cases of whitlow, of course, this tissue is involved, and this lesion has been already described. Distinct phlegmonous inflammation, apart from whitlow, is not as a rule observed. According to Arnozan, lesions of the peripheral nerves have only rarely as an effect the formation of ulcerative or gangrenous inflammation of the connective tissue. He cites Raynaud, and speaks of gangrenous phlegmons of rapid march which invade the lower limb as results of wounds of the sciatic nerve. But, as Arnozan states, the part played by the vascular lesions in these cases is difficult to distinguish from that played by the nervous lesions.

Pseudophlegmon may be described as that characteristic swollen and reddened state of the skin, sometimes quite painful, which follows irritative lesions of nerves. It has been referred to under the head of glossy skin. It does not lead to suppuration; certainly any case in which suppuration should occur would be exceedingly rare. Its appearance, however, simulates occasionally a beginning suppurative process. It was first described by Hamilton in a case of injury to a nerve. It is seen sometimes also in cases of trifacial and other forms of neuralgia.

Acute Decubitus.—This is a form of bedsore, rapid and malignant in type, which is not infrequently seen to follow lesions of the spinal cord. It occurs also in cases of cerebral lesion. It begins usually in a few days—sometimes within twenty-four hours—after the initial lesion, as, for instance, fracture of the spine with crush of the spinal cord. An erythematous spot first appears, then bullæ form; these contain a serosanguinolent fluid. Rapid destruction of tissue follows, until—as over the sacrum, for instance—immense deep-seated sloughs form and come away. The bone may be exposed and even involved. This lesion will not be described here in detail, because practically it is not seen in cases of peripheral lesions. Yet it cannot be doubted that in these cases of acute decubitus the peripheral neurons are extensively involved. It is probable that in one sense acute decubitus is thus due to a peripheral lesion, or at least that the trophic functions of the peripheral neurons are seriously impaired. As these extend into the cord, they are evidently involved in the central destructive lesion, in cases at least of injury to the spinal cord; but it is apparently essential that this central lesion should exist, because injury to the peripheral neurons outside of the cord, as in cases of injury to the nerve trunks, does not cause this type of bedsore. In cases of widespread multiple neuritis bedsores may form on the buttocks, heels, etc.; but these are not of this rapid and malignant type, and differ in fact in no way from the ordinary bedsore caused by pressure and uncleanliness.

Trophic Lesions of the Vascular System.

Lesions of innervation of the blood-vessels occur no doubt after nerve injury, but these cannot properly be called trophic lesions. Division of the sympathetic causes dilatation of the capillaries, but this is merely a paralytic and not a trophic phenomenon. A true trophic lesion is one in which the nutrition of the tissue is affected. The nutrition of the vascular tissue is no doubt affected, along with the other tissues, in such destructive lesions as whitlow, perforating ulcer, etc.; but whether this is primary or secondary is a question.

Distinct primary trophic lesions of blood-vessels—such, for instance, as would cause destructive changes in the walls of the vessels—are practically unknown.

Division of the cervical sympathetic nerves in young growing animals results, according to Sterling and to Bidder, in hypertrophy of the ear and increased growth of the hair on that side.

Alterations in the secretion of sweat may be included here. Irritation of the cervical sympathetic causes, among other symptoms, hyperidrosis; *per contra*, paralysis of the cervical sympathetic causes anidrosis. Neither of these phenomena, however, is constant or enduring. These phenomena will be described more fully under the head of Diseases of the Sympathetic System.

In affections of the abdominal sympathetic there may be increased or diminished secretion from the intestinal glands. This may be indicative of a genuine trophic lesion in the glands, due, however, rather to an affection of its nerve than to a true vascular affection.

Trophic Lesions of the Viscera.

It cannot be claimed that we have any very definite knowledge on the subject of trophic lesions in the viscera as results of either central or peripheral diseases of the nervous system. Brown-Séquard (see Arnozan[2]) announced that lesions of many parts of the cerebrospinal axis were followed after very brief intervals by hemorrhages into many of the viscera. Vulpian criticised these results, claiming that all such lesions in the viscera could readily be caused by emboli, and that such emboli could gain entrance by way of the wounds in the skull and spine caused in making the experiments. Certainly pathological anatomy in the human subject teaches but little if anything about such visceral hemorrhages following nerve lesions. Cerebral hemorrhage, it was taught by Ollivier, is frequently accompanied or followed by pulmonary apoplexy and by hemorrhage into other organs. While these phenomena may be seen in some cases, it would be rash to claim that they are in any sense "trophic" lesions. They simply represent diseased heart and blood-vessels, or septic processes, which acted primarily as the cause both of the cerebral and visceral lesions. The attempt to prove that such hemorrhages are more common after lesions of certain regions of the brain does not now find countenance. The same may be said of pneumonia following cerebral apoplexy. Cruveilhier is quoted by Arnozan as crediting the claim that pneumonia is a prompt and often fatal result of hemorrhage in the brain. These cases undoubtedly occur, but the pneumonia is evidently only a secondary result of the obstructed respiration and circulation in these cases. The lung lesion is usually more truly a

hypostatic congestion than a pneumonia. Fabre referred to "pneumonia of nervous origin," due to a lesion of the pneumogastric nerve, which lesion may be due to tumors of the mediastinum, œsophagus, or aorta. Such theories are rather fanciful—especially that of Fernet, quoted by Arnozan, that pneumonia is a form of *herpes*, which is a trophic lesion due to an inflammation of the pneumogastric nerve.

Hypertrophy of the heart as a result of moral affections was formerly claimed, but it is needless now to say that such a theory is no longer held, and that, even in case it were admitted, such an hypertrophy could not be regarded as a true trophic lesion.

Gastrointestinal lesions, especially the duodenal ulcer following extensive burns of the skin, have been held to be examples of trophic lesions (Brown-Séquard). The evidence to this effect is not satisfactory.

Obolenski cut the spermatic nerve and observed atrophy of the testicle. Arnozan, after citing a few clinical facts, inclines to believe that atrophy of this organ follows lesions of both the central and peripheral nervous system, but, as he says, the subject needs further study.

On the whole, it cannot be satisfactorily demonstrated as yet that many or grave trophic lesions occur in the viscera as results of affections of the nervous system.

Disorders of the Reflexes.

The physiology of the reflexes has already been described in a preceding section (page 34). Their pathological significance is now to be noted.

The reflexes may be either exaggerated, diminished, or abolished by disease.

They are exaggerated by those diseases that cut off the peripheral neurons from the inhibitory influence of the cerebral centres; consequently, as a rule, only in diseases of the spinal cord and cerebrum. For instance, any lesion gradually formed, or any sudden lesion after the influence of the primary shock has passed away, in any part of the cerebrospinal axis, provided the motor paths from the cerebral cortex are involved, will cause exaggeration especially of the *deep* reflexes. The commonest of these lesions are hemorrhage in the internal capsule, involving as it does the motor pathway; degenerative lesions in the pons or medulla, such as bulbar palsy; and the various destructive lesions of the cord, such as syringomyelia, meningitis, myelitis, lateral sclerosis, neoplasms, and traumata. In all such cases the deep reflexes that have their seats in the cord *below* the lesion are exaggerated. Such reflexes, however, as have their

origins at the *level* of the lesion, so that their motor neurons are involved in the destructive process, are abolished; and those that arise *above* the lesion are unaffected. Thus in a case of cervical lesion of the spinal cord, the reflexes in the arms may be abolished while those in the legs will be increased; but in a case of lesion of the lumbar spine the arm reflexes will be normal, while the leg reflexes will probably be diminished or abolished. This is simply because the reflexes arising at the seat of the lesion have their sensory or motor neurons, or both, impaired or destroyed; while those arising below the lesion have their neurons made more excitable by being freed from the inhibitory influence of the cerebrum. (For a full discussion of this and other theories of the reflexes, see page 34.) It follows from all this that exaggeration of the deep reflexes is almost invariably a sign of central disease. Such exaggeration does not arise in lesions of the peripheral nervous system, and this is because such lesions in the vast majority of cases impair the conducting power of the nerves. Exceptions to this rule, to be sure, have been claimed by some observers to occur in the initial stage of an irritative lesion of the nerves. For instance, it is said that exaggerated reflexes may be seen occasionally in the early stage of peripheral neuritis; this is claimed to be due to irritation, and consequently increased conducting power of the peripheral neurons. But it is certainly most rare, and I for one do not recall ever having seen it. Pressure on a nerve trunk, when this pressure is temporary and is caused for experimental purposes, has been found by Bastian, Vulpian, and others (see page 55) to cause sometimes cramps in the muscles supplied by the compressed nerve; but this is evidently due to direct irritation of the nerve trunk and is not a reflex phenomenon.

Abolition of the deep reflexes may be caused, under some circumstances, by shock. For instance, a total transverse lesion of the spinal cord, caused suddenly by accident, such as fracture of the spinal vertebræ, will cause abolition of the deep reflexes that arise even far below the seat of the lesion (Bastian). Thus a middorsal lesion of this kind will cause abolition of the knee jerks. So also will a large hemorrhage in the internal capsule, only in this case the abolished knee jerk is on the paralyzed side alone. In such cases, if the patient survive, the abolished reflexes will ultimately return and become exaggerated. This abolition of the reflexes below the seat of lesion, in cases accompanied with shock, is in apparent contradiction to the law already laid down; yet it is an undoubted clinical fact, and must be accepted as such. Its explanation is entirely hypothetical: the abolition is apparently due to shock, which is imparted to the neu-

rons that convey the reflexes, and in some way paralyzes them for a time. This abolition, due to shock, must evidently be carefully distinguished from the abolition which is caused by destructive lesions involving the so-called "reflex arc," *i.e.*, the peripheral neurons themselves.

The reflexes are always abolished or diminished by lesions which impair the peripheral neurons through which these reflexes pass. This is a rule to which there is no exception. Consequently this abolition or diminution is seen in all diseases of the spinal cord and nerves that involve the entering peripheral .sensory neuron (nerve trunk, posterior ganglion, posterior root, and posterior columns), and the cell body and axis cylinder of the peripheral motor neuron (anterior horn, anterior root, and nerve trunk). The most common of these diseases are neuritis in all its forms, locomotor ataxia, and the various degenerative and inflammatory affections of the anterior horns, such as anterior poliomyelitis, syringomyelia, and localized myelitis. In the last-mentioned diseases, of course, only those reflexes are abolished that have their course through the affected levels of the cord. It follows from this rule that abolition or diminution of the deep reflexes is a common result of all destructive lesions of the peripheral nervous system, but that it is not entirely confined to such peripheral diseases.

In cerebellar diseases the reflexes are variously affected, but the exact reasons for this are not at all clear. In cerebellar tumors the knee jerks may disappear and then reappear; this is probably due to variations in pressure beneath the tentorium.

The *superficial* reflexes are subject to practically the same laws that control the deep. They are, however, rather of positive than of negative value. In other words, their presence demonstrates that the nerves which conduct them and the segments of the cord through which they pass are healthy, but their absence is not always satisfactorily indicative of disease, unless it is confirmed by other symptoms. This is so of some more than of others, because the superficial reflexes are not all equally demonstrable in all healthy persons. The cremasteric reflex is one of the most common and constant, but, of course, is peculiar to the one sex. It varies, however, in healthy men. It is commonly abolished in lesions of the lumbar spine, but not so commonly in multiple neuritis. The plantar reflex is not readily demonstrable in some healthy persons, consequently its absence must be judged cautiously. The same may be said of many of the other superficial reflexes. The ocular and conjunctival reflexes, however, are constant in all healthy persons; consequently their diminution or absence is of positive value.

The anal and vesical reflexes are often involved in lesions of the lumbar region of the spinal cord, but not commonly in multiple neuritis. In middorsal lesions they may be exaggerated early in the case; consequently the bladder and rectum empty themselves by a truly reflex involuntary act. In destructive lesions of the lumbar cord these reflexes are abolished; consequently there is no reflex extrusion of the urine and fæces, but these escape simply because there is not sufficient resistance offered to them. This escape, however, often occurs only when the bladder and rectum are distended with their contents, and the evacuation is usually incomplete. In case of the bladder this constitutes the well-known "overflow" phenomenon, and is usually a sign that the viscus is distended with urine. This is also true of the rectum. In any isolated lesion that involved merely the nerves to the bladder and rectum, these reflexes would of course be abolished; but such isolated lesions of these nerves, without injury to the cord or cauda equina, must occur so rarely that they can practically be left out of account. I have never seen or heard of such a case.

The methods for testing the various deep and superficial reflexes have already been described in the section on their physiology.

To conclude, the two chief laws underlying the pathology of the reflexes, so far as the peripheral nervous system is concerned, may be briefly restated:

1. Exaggeration of the reflexes does not occur in diseases of the peripheral nervous system. Rare exceptions are noted.

2. Abolition or diminution of the reflexes occurs very commonly in destructive diseases of the peripheral nervous system, but is not confined to them, since it is seen in those diseases of the spinal cord that involve the course of the peripheral neurons; also in central diseases causing shock; also, for some obscure reason, in some lesions of the cerebellum.

Disorders of the Circulation.

In some disorders of the peripheral nerves, especially destructive lesions, such as neuritis and section, the circulation in the affected limb is obviously involved. The pulse rate is, of course, not affected, but the vascular tension is apparently somewhat impaired. This is seemingly due to involvement of the vasomotor system. Thus the parts become swollen, even œdematous, and the capillary circulation is evidently at fault. In some cases, in which symptoms of irritation of the nerves are present, the skin may be swollen and injected, presenting the phenomena of glossy skin or pseudophlegmon, already

described. In some cases of long standing the skin may be mottled, and the arm or hand may have a cold and lifeless appearance. Some of the trophic lesions already described are evidently due in part to, or at least promoted by, the weakness of the capillary circulation.

Alterations in the *temperature* of the parts may occur, and are evidently due to the impairment of the vasomotor system. According to Bowlby,[14] immediately after section of the median nerve the distal parts become suffused with blood and hotter to the touch. Mitchell demonstrated that compression of the ulnar nerve at the elbow is followed by a rise of temperature in its area of distribution, but this rise did not occur if a tourniquet had been previously placed on the brachial artery. This seems to prove that the elevation of temperature is due to a paralysis of the vasomotor system. Bowlby shows strikingly in a table the effect upon temperature of the division of the ulnar nerve. Eight hours after the accident the temperature, taken between the fourth and fifth fingers (supplied by the ulnar nerve), was 100.2° F.; while between the first and second fingers (which area is not supplied by this nerve) it was 99° F. This difference of about 1° remained constant despite alterations in the general temperature of the body. In long-standing cases, however, the temperature in the affected area falls remarkably, and the parts, as already said, are livid, mottled, and cold. Hutchinson[15] observed, in one of his reported cases, as great a fall as 16° as compared with the sound side. This decline in temperature seems to be coincident with the continuance of degeneration. As the nerve regenerates, the temperature gradually comes back to the normal. These facts prove conclusively that the activity of the circulation, and consequent activity of the nutritive functions, are much impaired by nerve wounds.

DISEASES OF THE CRANIAL NERVES.

The Olfactory Nerve.

The olfactory nerve, or properly nerve filaments, is composed of a series of peripheral neurons which, according to Jakob, are to be considered as analogous to those whose cell bodies are in the intervertebral ganglion. These special sensory cells are found in the epithelial layers of the olfactory membrane. The axis cylinder is an unmedullated fibre running to the olfactory bulb, where it splits up into its terminal arborizations about cells which compose the glomeruli olfactorii. These peripheral sensory neurons are analogous to the common sensory neurons in the earthworm, as demonstrated by Lenhossek. The cell body in the earthworm is lodged in the epithe-

lium of the skin, with a very short dendron but with a long axis cylinder connecting it with the central nervous organs. The cell bodies of the olfactory nerves are deeply pigmented, and this pigmentation is apparently closely identified with their physiology. Thus in the rare cases that have been observed in which absence of this pigment has been seen, abolition of the sense of smell has been the prominent symptom. Hutchinson,[55] of Kentucky, for instance, reported the case of a negro boy, who up to his twelfth year had a normal black skin. At this time a white patch appeared near the inner canthus of one eye. This patch gradually enlarged, so that in ten years the whole external surface of the patient's body was devoid of pigment. At the time that the boy began to lose the pigment in his skin, he began also to lose the sense of smell; hence the inference seems justifiable that this loss of smell was dependent upon a loss of the pigment in the epithelium of the nasal mucous membrane.

Althaus[56] relates the case of an albino who in early life had great difficulty in reading, and who late in life lost his smell and taste altogether. The inference in these cases is that the loss of pigment in the hair and skin and retina * is associated with a similar loss in the epithelium of the mucous membrane of the nose. The central portion of the olfactory apparatus, *i.e.*, the olfactory bulb with the olfactory tract, is not properly to be considered as part of the peripheral system. This olfactory bulb and tract constitute in fact a process of the cerebral hemispheres. They are covered with a cortex of peculiar structure, in which are lodged the cell bodies of the central neurons which communicate in turn with the brain cortex (Edinger). In the human being the olfactory lobe is much atrophied, and this is in accord with the well-known physiological fact that in man smell is a sense of secondary importance.

There can be no doubt that the function of the olfactory nerves is to convey to the brain the sense of odor. It has not always been considered so, however, for Magendie, according to Althaus, concluded from his experiments on dogs that smells were conveyed by the nasal branches of the fifth pair of nerves, and that the first nerve was a structure like the pineal and pituitary glands, the functions of which were as yet unknown. Bernard inclined also to this view. It has been objected justly to Magendie's experiments that they were made with ammonia and strong cheese, and that these substances irritated the endings of the fifth nerve. Hence the reactions which he observed in his dogs were not due properly to excitation of the sense of smell, but to irritation of the trigeminal nerves. Later physiological exper-

* The pigmentary layer is usually described by anatomists as part of the choroid; embryologically, however, it belongs to the retina.

iments, notably by Schiff and by Vulpian, have proved conclusively that the sense of smell is dependent upon the first nerve. It is important, however, to distinguish here between the sense for acid, bitter, sweet, and saline substances, and that for aroma or flavors. It is this last which properly constitutes the sense of smell, and it is this also which is the contribution, we may say, of the sense of smell to the sense of taste. Althaus has claimed that the olfactory nerve responds specifically to the application of electricity. This is in accord with the law of electrical reaction, and is seen also in the optic and auditory nerves. A current of great power is required to make an impression on the first pair, and this strong current so affects the other cranial nerves, especially the fifth, that it is somewhat difficult to distinguish the special effect upon the olfactory nerve. In Althaus's patient, however, there was complete paralysis of the fifth pair, so that many of these disturbing influences were removed. In this case a distinct perception of a phosphorus smell occurred on the application of a current of thirty-five pairs of plates.

For the proper action of the olfactory nerves the mucous membrane of the nose should be moist; hence anything that checks the proper secretion of mucus in the nose will interfere with the conduction of olfactory impressions along these sensory neurons. Excessive secretion, on the other hand, especially when associated with swelling of the mucous membrane, will also interfere with the proper action of this nerve.

Distinct or isolated diseases of the olfactory nerves are exceedingly rare. The function of the nerve is sometimes apparently abolished or suspended in hysteria, but as this is a central disease it cannot properly be claimed that the peripheral neurons are involved in such cases.

The olfactory nerve is said to be sometimes congenitally absent (Althaus). Whether this claim refers to the peripheral neurons proper or to the olfactory bulb and tract, is not plain. Braschet, quoted by Althaus, has seen this congenital anosmia in several members of the same family, but his observations do not seem to have been founded upon anatomical studies. Loss of pigment in the body generally, and in the olfactory epithelium of the nose especially, has, as already said, been associated with a loss of smell.

Inflammation of the olfactory nerve proper is probably of extremely rare occurrence. Althaus calls attention to the singularity of this fact when we consider the frequency of optic neuritis. The latter, as is well known, is very common with diseases of the brain. The analogy, however, between optic neuritis and an olfactory neuritis under these circumstances is not very apparent, from the fact that the

connections of the olfactory neurons with the brain are not so extensive and the course of these neurons is not so long as in the case of the optic nerve. Althaus reports a case of apparent olfactory neuritis occurring in a banker's clerk after exposure to cold. He had the symptoms of a peripheral neuritis, such as numbness in his feet, and about the same time he perceived a strong smell of phosphorus, which persisted for six weeks. At the end of that time he had entirely lost the sense of smell. The perception of odors, such as of camphor, valerian, and ether, was abolished, but the patient experienced the usual sensation of tickling, choking, and lacrymation when ammonia was presented to the nose, and snuff made him sneeze; hence it was apparent that the fifth nerve was not involved. This patient's taste for flavors and aroma was also abolished, but not that for sugar, salt, quinine, and acid. This fact proved that the sense of odor is an integral portion of the sense of taste. The patient died, and an autopsy by Ferrier revealed the naked-eye appearances of neuritis of the first pair at the base of the brain.

Degeneration of the olfactory nerve filaments is said to occur in some cases of locomotor ataxia and other degenerative diseases of the central nervous system (Herter). It is possible that such instances would be reported more frequently if greater care were taken to examine the first pair in such diseases.

Injuries to the head are undoubtedly the commonest cause of impairment to the first pair. Ogle[80] has collected several of these cases. Blows upon the head are undoubtedly the commonest of these injuries. In two of Ogle's cases the blow which caused the anosmia was on the occiput. Ogle calls attention to the fact in this connection that a blow at the back of the skull is not nearly so likely to injure the part of the brain immediately underneath as it is to injure the anterior and inferior portions where the olfactory nerves join the brain. The reason for this is that the anterior brain rests directly upon the bones of the skull. A blow which is not sufficient to injure the anterior brain generally may still suffice to tear the olfactory nerves, owing to their small size and their delicacy of structure.

Tumors and other gross lesions at the base of the brain or about the bones of the nasal cavities, and chronic catarrh of the mucous membrane, may cause injury to the olfactory nerves and loss of smell. Such lesions are apt to cause other symptoms which are still more prominent than the anosmia and may even disguise it.

Excessive stimulation may destroy temporarily or permanently the sensitiveness of the olfactory nerves. Cases are reported of scavengers and workmen in cesspools being thus deprived of the sense of smell. Graves reported the case of a soldier who was en-

gaged in tearing out an old cesspool and was thus exposed for a long time to an overpowering stench. He entirely lost his sense of smell, and thirty-six years afterwards the anosmia persisted. Althaus suggests that such cases may possibly be owing to capillary hemorrhages in the nerves.

Hyperosmia is occasionally seen in cases of insanity, hysteria, and epilepsy, but is probably a central rather than a peripheral affection.

The *treatment* for all these conditions is not very satisfactory. In surgical cases, in which the injury to the olfactory nerves is direct and severe, little if anything, of course, can be done by medical treatment. Strychnine, when applied directly to the nasal mucous membrane, as in the form of a snuff, is said to increase the keenness of the sense of smell. This is so to such an extent that snuff, ammonia, and other active agents cause positive pain. This is due probably in part to stimulation of the fifth nerve. Morphine on the other hand, is said to deaden the sense of smell. In the hyperosmia of epileptic patients Althaus injects one-sixth of a grain of acetate of morphine with one-sixtieth of a grain of sulphate of atropine subcutaneously. He claims that this acts satisfactorily as a sedative.

Electricity may be employed in some cases of anosmia in the hope of stimulating the olfactory nerve filaments. This agent, however, does not cause a satisfactory reaction in these filaments unless a strong current be used, and such currents about the head should be used very cautiously.

The Optic Nerve.

The optic nerve and its nervous expansion in the eyeball, the retina, are usually considered by clinicians as pertaining to the peripheral nervous system. Morphologically, however, this view is incorrect. The optic nerve and retina are really formed embryologically by processes from the first cerebral vesicle. While the diseases of this nerve will be included here in this paper among peripheral diseases, it is necessary, for a correct understanding of the anatomical relations of the nerve, to study it briefly from the embryological standpoint.

Very early in embryonal life two processes are budded out from the primary brain vesicle. Each of these processes forms at its end a cup-like cavity which remains in connection with the brain proper by a stalk. This cup-like cavity is formed by an infolding of the epiblastic tissue. As a consequence of this infolding the optic vesicle presents two distinct layers. The outermost of these layers is much thinner than the innermost. This outer layer soon shows a

deposit of dark pigment and forms the future pigmentary layer of the retina. The innermost of these layers is much thicker and forms the retina proper. Within this retina are developed those numerous and complicated nervous structures which will be briefly described.

The optic vesicle is the foundation of the eyeball or globe. This optic vesicle includes in its open cuplike orifice a round body, which forms the crystalline lens. The other structures of the eye, as the choroid, iris, cornea, etc., are formed from mesoblastic tissue. The history of their complex development need not be followed here. According to Piersol[11] this nervous tunic of the eyeball, regarded in the light of the new views of the retina as based upon investigations of Tartuferi, Golgi, Cajal, Dougiel, Retzius, His, and others, cannot be placed in the same category with the remaining coats of the eye, but must be conceded a morphological position of far greater dignity and importance. Piersol says truly that the retina is a true nervous centre, and that it is really a portion of the central nervous system situated at the periphery. Following this author we can regard the retinal tract or tunic as extending from the entrance of the optic nerve posteriorly as far forward as the pupillary margin. As already said, the retina proper consists of an inner and an outer lamina and these represent the unequally developed layers of the original optic vesicle. The outer lamina constitutes the pigment layer, which is usually described as belonging to the choroid but which, according to embryology, must be regarded as part of the retina proper, all being formed from epiblastic tissue. To the inner or thicker lamina belong all the other layers of the retina. The inner layer is further subdivided by Schwalbe, quoted by Piersol, into the neuroepithelial and the cerebral layer. In order to understand these anatomical elements, which are of great importance from the standpoint of the neuropathologist, they may be tabulated, as has been done by Piersol, as follows:

I. Outer layer of optic vesicle.	Pigment layer.....	A. Pigment layer.
II. Inner layer of optic vesicle.	Layer of rods and cones. Layer of bodies of visual cells (outer nuclear layer).	B. Neuroepithelial layer.
	External plexiform layer (outer reticular layer). Layer of bipolar cells (inner nuclear layer). Internal plexiform layer (inner reticular layer). Layer of ganglion cells. Layer of optic nerve fibres.	C. Cerebral layer.

These elements, as in the case of the whole nervous system, may be divided into the nervous elements proper and the supporting ele-

ment. The latter in fact is a true supporting neuroglia, which appears as a reticular framework composed of columnar segments along the fibres of Müller. These extend through the entire thickness of the retina and constitute the structures known as the external and internal limiting membranes. For our present accurate knowledge of the retina we owe much to Golgi's silver stain and to the researches of Ramon y Cajal. The importance of these observations, as pointed out by Piersol, lies especially in the establishment of the broad theorem so often advocated in this paper of the independence of nerve cells and their extension as axis cylinders. In the retina as in all other portions of the nervous system the nerve cell is an independent anatomical unit and does not end in a common network or by actual union with other cells. As in other portions of the nervous system, the distinction holds also in the retina between a set of peripheral neurons and a set of central neurons. Only in the case of the retina, as will be seen, these peripheral neurons have exceedingly short courses and are included almost entirely in what may be considered the peripheral structures of the retina.

The first or outer layer of the retina, as already said, must be considered the pigment layer. Next come two strata which constitute the neuroepithelial layer. The first of these is the layer of rods and cones, the latter the external nuclear layer. These rods and cones are the visual cells proper. The rods terminate in little knobs, which are embraced by the arborization of the bipolar nerve cells of the outer ganglionic layer. The cone cells terminate by expansions, which are in contact also with the bipolar cells. These bipolar cells are the true peripheral sensory neurons of the retina—in other words, they constitute properly the peripheral nervous system of the organ of sight. They are not of equal length but they extend to various levels in the layers of the retina. The central expansions of these bipolar ganglionic cells come ultimately into close relations with the deeper seated set of nerve cells, which constitute properly the central sensory neurons. These central sensory neurons send out in turn long axis cylinders which go to form the optic nerve.

In addition to these elements in the retina Cajal has demonstrated certain horizontal or basal cells in the external plexiform layer, the processes of which ramify about the individual cells. The function of these has not been perfectly demonstrated. It is thus seen that the optic nerve is developed by the growth of the axis cylinders of the nerves of the deeper ganglionic layer in the retina. In the embryo these grow brainward, taking their course through the optic stalk, which has already been described as a part of the primary optic vesicle. As Ryder[1a] has pointed out, the optic nerve is thus devel-

oped independently of the optic stalk—in fact, the optic stalk serves simply as a tract or pathway to conduct these growing embryonal axis cylinders towards the points of their termination in the brain. The optic stalk, according to Ryder, is broken down in the course of development. According to Cajal's researches, in addition to these axis cylinders growing centrad, certain other fibres also exist growing from the brain into the retina. The course of the true optic fibres through the optic nerves and optic tracts is well known and easily demonstrated.

The course of the optic fibres from the retina to the brain may be divided into three sections, namely, the optic nerve, the optic chiasm, and the optic tract.

The optic *nerve*, leaving the eyeball, passes backwards through the orbit and gains entrance to the skull by the optic foramen. Its relations to the other nerves of the orbit are briefly as follows: It lies to the inner side of the third nerve and just above that nerve. It is crossed by the fourth nerve and by the ophthalmic division of the fifth. The sixth nerve passes well to its external side. Within the orbit the nerve lies freely embedded in connective tissue, fat, and the various muscles, nerves, and blood-vessels of the part. It leaves the orbit by the optic foramen in the sphenoid bone and passes at once into the optic chiasm.

The optic *chiasm* lies upon a narrow groove, called the optic groove of the sphenoid bone. This chiasm, or commissure, is formed by the junction of the two optic nerves. It is consequently the meeting place and the point of various courses and departures of the fibres of the optic nerve. The chiasm is flat, somewhat quadrilateral in form, or of the shape of a letter X. The fibres of the optic nerve within it are arranged as follows:

The fibres from each nerve entering the chiasm pursue three distinct courses. A few pass directly across and into the optic nerve of the opposite side. These evidently unite the retinæ of the two eyes. The larger portion of the optic fibres decussate with their fellows of the opposite side and pass on into the optic tract of the opposite side towards the brain. The third set of fibres continues into the optic tract of the same side. The chiasm contains, moreover, a fourth set of fibres, which pass from one tract to the other, apparently connecting the visual centres of each side of the brain. It thus appears that the chiasm is the meeting place of four sets of fibres, three of which are axis cylinders of the central sensory neurons of the retina. The importance of this arrangement of fibres in the chiasm cannot be underestimated from a pathological and diagnostic standpoint.

The optic *tract* is in reality only the continuation backwards of the fibres of the optic nerves. From the arrangement of these fibres in

the chiasm, however, it has now been made apparent that the optic tract does not contain identically the same fibres as the optic nerve of the opposite side, but that each tract contains fibres, first from the retina of the same side, second from the retina of the opposite side, and third from the other optic tract; therefore the optic tract is not to be considered as a mere continuation of the optic nerve of the opposite side. Leaving the chiasm, the optic tract passes backwards in the form of a flattened band and winds across and around the under surface of the crus cerebri. In its course it divides into two branches, which conduct its fibres to the corpus geniculatum, the corpora quadrigemina, and the optic thalamus. Within these parts these visual sensory neurons terminate by aborizations around ganglionic cells, which form various nuclei or pass backwards towards the visual centre of the occipital lobe of the brain. The main point of distribution of the optic nerves, however, is, according to Edinger, the roof of the midbrain, in those eminences called the corpora quadrigemina. Edinger really calls the quadrigeminal bodies the point of *origin* of the optic fibres. But our more recent knowledge negatives this view. The *origin* of an axis cylinder must be in its cell body, *i.e.*, in this case in the retina. The fibres end here apparently in a fine network formed of their arborizations around numerous delicate cells. The further minute anatomy of the optic tracts within the brain does not concern us here, as we are engaged in the study of the diseases of the optic nerve proper.

The end of the optic nerve at its junction with the retina and the retina itself may be subjected to direct visual examination. In this respect this nerve offers an opportunity to the clinician that is not found in any other nerve of the body without dissection. Upon inspection by the ophthalmoscope the eye ground, as it is called, presents a definite panorama. The most conspicuous object in the eye ground is the optic disc. This is the commencement of the optic nerve proper. It is a small somewhat oval disc, of lighter color than the surrounding eye ground and situated towards the inner or nasal side. Radiating from about the centre are seen the blood-vessels of the eye ground. These consist of an entering artery and an emerging vein. The artery is smaller in size and less tortuous than the vein. It also differs from it in color, being of a paler hue. It divides upon entering the eye into a number of branches, principally ascending and descending. These can be traced far out upon the eye ground proper, gradually dividing and subdividing into innumerable branches. The central portion of the eye ground is less richly supplied with these branches, than are the upper, lower, and inner segments. The optic vein is larger and of a deeper color and more tortuous than the

artery. Its area of distribution corresponds closely with that of the artery. As its numerous branches converge towards the disc they unite and pass outwards, disappearing from view in the small cup-like depression at the centre of the disc. Around the borders of the optic disc is usually seen a more or less complete and distinct rim of pigment. This is due to a slight exposure at this point of the pigmentary layer of the retina. The eye ground itself is of a bright pinkish or red hue, somewhat stippled in appearance.

The optic disc forms really a slight papillary eminence, hence called sometimes the optic papilla. It is composed, as already said, largely of the converging axis cylinders of the central sensory neurons emerging from the deep ganglionic layer of the retina. These pass out at the disc, being closely bunched together as it were, hence forming the papilla. Where they pass outwards from the disc their aggregate mass at once constitutes the commencing optic nerve.

In the centre of the disc a deep cup-like depression exists, called the physiological excavation. This results from the disposition of the emerging axis cylinders, which as they reach the disc bend and dip down in large bundles more or less close to the periphery. In the centre of the optic nerve are seen the retinal vessels already described.

The physiological excavation, so called to distinguish it from that produced by a pathological process, is usually in the centre of the disc. It is deeper in some eyes than in others, and in those cases in which it is deeper the walls are steeper. On longitudinal section of the optic nerve and retina at their point of junction, the parts present the following appearance. The choroid and retina end abruptly in order to form the orifice through which the nerve fibres escape. The sclera, or outer coat of the eye, apparently becomes continuous or blended with the outer sheath of the nerve, which is truly a prolongation of the dura mater. Within this outer sheath are the arachnoidal and pial sheaths, the former extending as a fold almost to the point of emergence of the nerve. Some fibres of the scleral coat, constituting a sort of feltwork or lattice work, interlacing with the nerve fibres, extend across and through the optic nerve just after its emergence from the disc. These constitute the lamina cribrosa. These fibres, or trabeculæ, support blood-vessels, so that by this arrangement the optic nerve at the base of the papilla has an exceptionally rich vascular supply. These vessels constitute an important communication between the ciliary and retinal vessels.

On transverse section the optic nerve is seen to be composed of a large number of bundles of nerve fibres. Each one of these bundles contains a large number of medullated nerve fibres. These

bundles are closely packed together, and are held by an intrafascicular connective tissue which also supports numerous blood-vessels. This tissue is a prolongation of the pia mater. The outer sheath of the optic nerve is a prolongation of the dura mater. Its inner sheaths are likewise prolongations first of the arachnoid and then of the pia. These enclose lymph spaces.

The optic nerve, according to Monro,[*] must be regarded, as already explained, as part of the central nervous mass, being developed not as an ordinary peripheral nerve but from an outbudding of the primary forebrain. Monro, in fact, regards it as comparable to the posterior column of the spinal cord. This is an important theorem from a pathological as well as an anatomical standpoint, because this nerve is extremely likely to undergo degeneration in some of the diseases that involve the spinal cord.

The entire number of nerve fibres within the optic nerve, according to Piersol, has been variously estimated. The estimates of Salzer and of Krause place this number at about four hundred and twenty-five thousand. Piersol also calls attention to another evidence of the close analogy existing between the retina with its stalk and the central nervous system. This is found in the fact that the axis cylinders in the former, just as in the latter, are devoid of a true neurilemma, the medullated fibres being held in place by neuroglia or supporting substance. The investigations of Cajal and others show conclusively the presence of large neuroglia cells within the supporting tissue of the optic fibres. The axis cylinders of the optic nerve, as already stated, are medullated; but they do not receive this white substance of Schwann in the retina, but only on emerging through the lamina cribrosa. It is this fact that accounts for the increased size of the optic nerve immediately after its emergence from the eyeball. The nodes of Ranvier are wanting.

In considering the physiology of conduction of visual impressions along the optic nerve and optic tracts it is necessary to pay especial attention to the course of the optic fibres within the chiasm. Each optic tract contains fibres not alone from one eye but from half of the retina of each eye, and these halves are on the same side of the eye as the individual optic tract. For instance, the right optic tract contains fibres from the right half of the retina of the right eye and from the right half of the retina of the left eye. These two halves, however, are not equally represented in each tract, because the decussating fibres are more numerous than the non-decussating fibres, therefore each optic tract represents a larger portion of the retina of the opposite eye than of the eye on the same side. Thus the right optic tract, conducting fibres from the right

or nasal half of the left retina, represents a larger field in that retina than it does in the right retina, in which it represents a smaller field on the right or temporal side. The reason for this is that the nasal half of the retina covers a larger field than the temporal half, and this is because the nose tends to limit the range of vision towards the median line of the body.

As a consequence of the anatomical relations of the optic fibres in the chiasm, lesions of the optic nerves, of the chiasm, and of the optic tract give different effects. These effects are as follows:

A total transverse lesion of the optic nerve causes complete blindness of the eye on that side.

Lesions of the chiasm produce various effects according to their location and extent. A lesion of the anterior portion of the chiasm, if it involves the decussating fibres from each eye; will cause blindness in the nasal half of the retina of each eye, but as this half of the retina corresponds to the opposite visual field, the blindness will be in each temporal field. In making this distinction it is necessary to recall that the nasal half of the retina represents the temporal half of the visual field; and *vice versa*, the temporal half of the retina represents the nasal half of the visual field. Consequently a lesion of the anterior portion of the chiasm, just referred to, produces bilateral temporal hemianopsia. Again, a small strictly limited lesion of one side of the chiasm, involving as it would only the fibres from the temporal half of the retina of that side, would produce blindness of that half only. For instance, such a lesion on the right side would produce blindness of the right or temporal half of the retina of the right side, which corresponding to the nasal half of the visual field would be a right unilateral nasal hemianopsia; *vice versa*, such a lesion on the left side would produce a left nasal hemianopsia. Again, a lesion of the posterior and median portion of the chiasm would intercept the decussating fibres from the nasal half of each retina and would consequently produce blindness in the temporal halves of each visual field; in other words, a bilateral temporal hemianopsia, just as has been described for a lesion intruding upon the chiasm from in front.

Binasal hemianopsia, by which is meant a blindness in the temporal half of each retina with corresponding loss of vision in the nasal half of each field, is a very rare condition. Veasey[92a] has recorded such a case in a woman with obscure nervous symptoms, and with eye-grounds somewhat suggestive of Bright's disease. He also reviews the literature, from which it appears that the pathology of the condition in all cases has not been satisfactorily determined. In one case a tumor of the third ventricle was found, and in another a tumor of the cerebellum. In the latter case, however, a meningitis

also was found about the chiasm. This rare form of hemianopsia probably always depends upon a bilateral lesion of the optic nerves (in front of the chiasm) involving the fibres to the temporal halves of the retinæ. Such a lesion may be due to syphilis. If it occurs in cases of brain tumor we must believe that the optic neuritis caused by neoplasm may be only partial in each nerve and may be symmetrically distributed.

Finally a totally destructive lesion of the chiasm involving all the fibres from both eyes would produce blindness in both eyes, namely, bilateral total anopsia.

A total destructive lesion of the optic tract produces still different effects. As this tract conducts fibres from the same half of each eye, the blindness would be in the half of each retina on the same side as the visual tract affected. Hence a lesion of the right optic tract would produce blindness of the right half of the retina of the right eye, and of the right half of the retina of the left eye; consequently the left half of the visual field for each eye would be affected. This is called a left homonymous hemianopsia; *vice versa*, a lesion of the left optic tract causes a right homonymous hemianopsia.

Congestion of the Optic Nerve.

Pathology.

The diseases of the optic nerve may be classified as congestion, primary atrophy, and inflammation.

The *symptoms* of congestion of the optic nerve visible by the ophthalmoscope are an increased color and a swelling of the disc. This color is best described as brick dust. The swelling causes an obliteration of the distinct outline of the disc, so that it is difficult to distinguish actively its margin from the general color of the eye ground. The veins also are unusually full and tortuous. This condition is sometimes difficult to distinguish, and its value as a clinical symptom can usually be determined only by an expert ophthalmologist.

De Schweinitz gives the following as the chief *causes* of congestion: (*a*) Errors in refraction, especially hypermetropia and hypermetropic astigmatism; (*b*) Occupations which expose their subjects to intense glare and heat; (*c*) Toxic agents, as tobacco, alcohol, and lead; (*d*) Some diseases of the eye, as, for instance, inflammation of the iris; (*e*) Disorders of the brain and spinal cord; (*f*) Heart disease, violent cough, and any cause that produces engorgement of the brain, as, for example, epileptic fits.

With reference to these several causes it may be said that dis-

orders of the brain and spinal cord are among the most important. Observations have been made especially in the various forms of insanity. Opinions differ, however, as to the frequency or importance of congestion of the optic disc in insanity. Some authorities have even failed to observe it at all. Albutt, quoted by De Schweinitz, has found the discs hyperæmic in mania, but sometimes the opposite condition, anæmia of the discs, has been observed. Albutt noted congestion more frequently in chronic dementia. Lautenbach,[83] in an extensive study of more than seven hundred insane patients in various forms of insanity, claimed that he had noted congestion of the retina in as many as forty per cent. of his acute cases, but this percentage is probably far higher than would be established by all observers. In some conditions of simple brain disorder in which atrophy of the optic nerve is finally established a preceding stage of hyperæmia has been observed by Albutt and others. De Schweinitz, however, failed to observe true congestion in these cases, although he says that there is not infrequently a dull-red appearance with marked grayness of the deeper layers. He has also seen such hyperæmia in focal lesions of the brain, as for example in cerebral embolism.

In chronic epilepsy Oliver[84] has observed congestion not so much as a low type of retinitis. In all conditions in which it is claimed congestion of the optic disc is a symptom the utmost care and expert knowledge are requisite to determine its true pathological importance. This function belongs rather to the ophthalmologist and the subject is best treated of in text-books especially devoted to the subject.

The *prognosis* will of course depend upon the exciting cause. If this can be remedied or relieved the congestion will disappear. In forms of chronic insanity and epilepsy the symptom is rather curious than important. It throws but little if any light upon the gravity of the case or upon its future course. In cases which are followed by atrophy the prognosis of course becomes graver, because this atrophy is probably coincident with and significant of degenerative changes in the brain.

Treatment of congestion of the optic disc as an isolated symptom in diseases of the brain and spinal cord is scarcely called for. The condition when excited by temporary causes will disappear without treatment. When it is excited by recurrent causes no treatment will likely avail either to prevent or relieve it. When excited by refractive errors or by intense glare and heat, rest of the eye and relief from the exciting cause are all that will be required.

Optic Neuritis.

Inflammation of the optic nerve is a most important symptom to the neurologist. While it usually is assigned to the province of the ophthalmologist it should not be forgotten that it is essentially a neurological phenomenon, that it throws most important and often essential light upon numerous diseases of the nervous system, and that in consequence it is properly included in the study of nervous diseases. It is the one instance in which the neurologist is able to see a disease process in the living nerve. Consequently it presents to his eye a most realistic picture of disease. He knows at once that it is significant of still deeper degenerative or inflammatory processes in the central nervous system. It is consequently a symptom which never misleads, but the failure to observe it may often result in the most grave errors in diagnosis.

Optic neuritis has been divided by many writers into various classes, such as neuroretinitis, descending or interstitial neuritis, and choked disc. It is best, however, for our purposes here to treat of it under the simple designation of optic neuritis or papillitis. It is proper to say, however, that the distinction between descending neuritis and choked disc may have practical importance. The difficulty, however, is in determining this distinction. Choked disc as described by von Graefe is that condition of engorgement, swelling, and mechanical obstruction which is supposed to depend upon lesions of the brain which interfere with the return circulation through the retinal veins. Descending neuritis, on the other hand, as the name implies, is an active inflammatory process extending along the optic tract and optic nerve from a centrally located lesion. In practical work, however, it is seldom possible to distinguish satisfactorily between these two conditions. Choked disc is evidently soon associated with inflammation and degeneration of the axis cylinders as they emerge from the eyeball, while, on the other hand, a descending neuritis is accompanied by swelling of the disc and engorgement of the vessels, which practically constitute a choked disc. It is rather the opinion now of ophthalmologists, following Hughlings Jackson, that there is but one kind of optic neuritis, and that the one element of swelling of the disc varies in different cases according to the exciting cause.

Various theories have been advanced to account for optic neuritis in diseases of the central nervous system. Von Graefe's theory that it was caused by increased intracranial pressure, while it may be true in some instances, as in gross lesions, brain tumor, etc., is not

adequate to explain it in many other instances. The same may be true of the theory which ascribes neuritis to an obstruction of the lymph spaces. The theory advanced by Jackson, that optic neuritis may be due to an intermediation of the vasomotor nerves, is exceedingly obscure and unsatisfactory. The most probable theory, it seems to me, is that in many instances the sensory neurons of the optic nerve suffer by the direct action of some irritating material in the blood. This we know is the mechanism by which a neuritis is set up in all other parts of the body. Obscure theories of mechanical or vasomotor intervention are scarcely necessary. The view, on the other hand, that these optic neurons in many diseases, as for instance locomotor ataxia, paretic dementia, lead poisoning, are affected by the direct action of the same causes that produce the other characteristic lesions in the nervous system (*i.e.*, the circulation in the blood of some poisonous material such as that of syphilis or lead) is the most rational. This view is curiously supported by the teachings of embryology. Monro, as already stated, regards the optic nerve not as an ordinary peripheral nerve but comparable to the posterior column of the spinal cord. Hence it is reasonable to suppose that it would be subject to the same diseases from the same causes that occur in the central nervous system. Hence also the direct ravages of syphilis and of chronic toxæmias may occur in this nerve as in other tissues of the cord or of the brain. It is not so easy perhaps to understand why optic neuritis is such a common accompaniment of brain tumors. The theory of toxæmia in these cases seems scarcely warranted. It is noteworthy, however, that this symptom occurs most commonly in cases of tumor about the base of the brain, and especially beneath the tentorium. In such cases it may be that the theory of an obstruction to the vascular or lymph systems may be the correct one. It is conceivable, for instance, that obstruction of the lymph spaces might lead to the damming up of deleterious products and that these, undergoing retrograde changes, might act by interfering with the nutrition of these sensitive neurons or by directly poisoning them. It must be remembered too that foci of infection may occur in cases even of tumor of the brain. For instance, the interference with the nutrition of the brain mass may so lower the vitality of parts that they may readily suffer from various kinds of toxins. Cases have been observed, in fact, in which a direct connection has been established between inflamed optic nerves and some area of inflammation or degeneration in the brain. This is rendered all the more easily understood when it is recalled that the central connections of the sensory optic neurons are very extensive and numerous.

The *symptoms* of optic neuritis are both subjective and objective.

The subjective symptom is impairment of vision. This, however, is not always present; in fact, a high grade apparently of swelling and inflammation may be present without the patient being conscious of any limitation of his sight. In such cases, however, expert tests will often demonstrate that the fields of vision are contracted or otherwise limited. In cases which are far advanced, however, sight usually becomes impaired, and in many cases may become ultimately totally

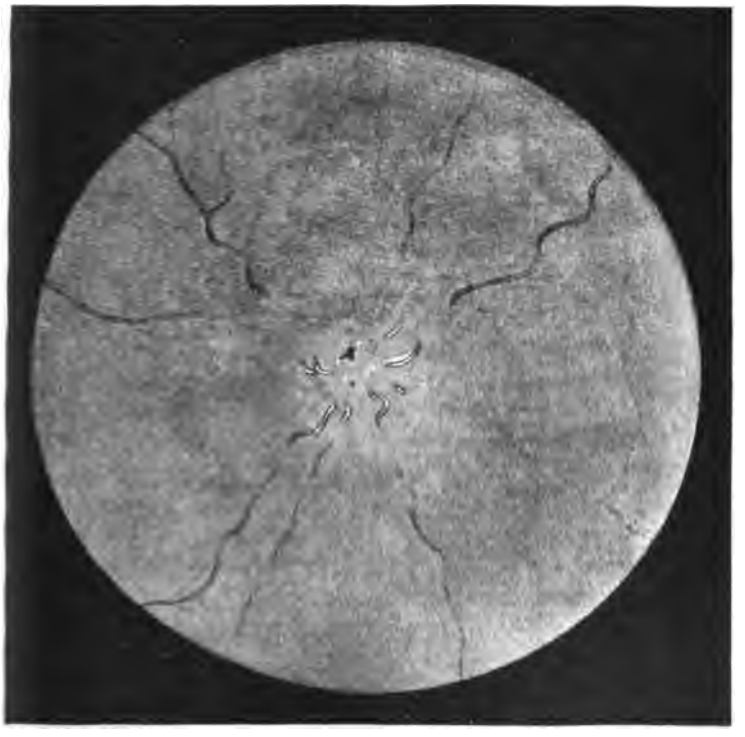

FIG. 15.—Optic Neuritis Associated with Tumor of the Cerebellum.

abolished. Blindness is occasionally an early symptom of optic neuritis and may even occur almost suddenly. These variations are no doubt due to variations in the completeness of the destructive process in the neurons and especially to the number of neurons involved. These factors will of course differ in different cases. Pain is not a symptom of optic neuritis. If present in these cases it is due to the central lesion, such as tumor or abscess, which causes the neuritis. The field of vision may be variously impaired. It may be concentrically contracted or some segment may be more involved than others. True hemianopsia is not caused by pure optic neuritis. When it oc-

curs in such cases it is due to a unilateral lesion somewhere in or behind the optic tract. Perception of colors is sometimes affected by optic neuritis; that of red and green, according to De Schweinitz, being usually lost first. The pupils are not affected until blindness occurs, when of course the reaction of the iris to light is lost.

The objective symptoms of optic neuritis are visible only with the aid of the ophthalmoscope. They are highly characteristic. The most common of these symptoms is swelling with increased vascularity of the papilla. This causes the outline of the disc to become obscure until eventually the edge or rim is lost to view entirely. A very early stage of this swelling, before the edge of the disc is obscured, may be difficult to determine. The first change, according to Gowers, is noted on the nasal side. This is due probably to the fact that there are more nerve fibres on the nasal than on the temporal side. As the change advances, however, the whole disc becomes obscure and the physiological cup is filled up and obliterated, although some evidence of it, as a very slight depression, may remain visible. The color of the swollen papilla is redder than normal as a rule, and it frequently has a striated appearance, due to its increased vascularity. As the swelling increases it extends out somewhat over the retina, so that its diameter may be two or three times as great as the diameter of the normal disc (Gowers). White blotches are seen on its surface. As the swelling increases the veins become much swollen and more tortuous, while the arteries are distinctly narrowed. Under the microscope the swollen papilla shows distended blood-vessels and capillaries with an accumulation of leucocytes in the tissues and especially in the perivascular sheaths. The nerve fibres are seen to be degenerated just as in neuritis in other nerves. Globules of myelin are noted. The products of degeneration are seen, and the walls of the arteries are thickened. The sheath of the nerve behind the globe is distended so as to form a pyriform swelling.

The *causes* of optic neuritis are numerous and may be classified under the two heads of focal nervous diseases and general disorders.

Brain Tumor.—Among the most common causes of optic neuritis is brain tumor. This is such a common symptom of intracranial growths that an ophthalmoscopic examination in suspected cases is of the first importance. I have known several instances of incipient brain tumor in which the detection of inflammation of the optic papilla was the first convincing proof of the presence of a tumor. Of the one hundred cases tabulated by Mills and Lloyd [6] choked discs were recorded in eight and optic neuritis or neuroretinitis in eighteen. This gives a percentage of twenty-six. The distinctions, formerly insisted upon

by Albutt, Norris, and others, between choked discs, papillitis, and congestion are not observed in this general statement. As said already, these distinctions are no longer considered of great diagnostic importance. The general result in these one hundred cases shows that the percentage of optic neuritis in brain tumor is so high that this symptom must be regarded as of first importance. According to this table the optic nerves were involved in tumors situated in all portions of the encephalon. They were not infrequently present in tumors of the convexity. Descending neuritis, however, according to these authors, would seem likely to occur when the tumors are so situated that inflammation set up by them can readily extend by anatomical continuity along the membranes to the optic nerves.

The relative preponderance of optic neuritis in tumors in various regions of the brain is difficult to estimate; so too is the absolute percentage in all cases. The percentage given above from Mills and Lloyd's table is far below that which is usually claimed by neurologists. This may be accounted for by the fact that that table was based on cases picked at random from medical literature, and that probably ophthalmoscopic appearances were not noted in all of them. According to Gowers[66] and Bramwell [67] optic neuritis is present in at least eighty per cent. of all cases, and this, according to Oliver,[68] is probably correct, but Nuske and Reich, quoted by Oliver, claim a still higher ratio. With reference to the location of the tumor, it is not possible to formulate a positive law. The general statement, however, is permissible that tumors about the base of the brain, and especially beneath the tentorium, are much more liable to cause optic neuritis than tumors about the convexity. Again, tumors of the parietal and especially of the occipital lobes, impinging as they may upon the optic fibres and optic centres in the brain, are still more liable to cause optic neuritis than tumors of the frontal lobes. I have known several instances in which tumors of the frontal lobes and one in which a tumor in the Rolandic region did not cause an optic neuritis. These facts seem to indicate that obstruction of the vascular system or of the lymph spaces is at least one factor in the causation of optic neuritis. Tumors in the frontal or antero-frontal region are the least likely of all brain tumors to cause pressure on the optic nerves or on the vessels at the base of the brain. The products of retrograde metamorphosis in such cases would probably be taken up and removed without the same risk of infecting the optic nerve fibres. Tumors of the cerebellum are undoubtedly the most prone of all brain tumors to cause optic neuritis. So true is this that it may be laid down almost as a law that optic neuritis is seldom if ever absent in any case of cerebellar tumor during its entire course.

According to Oliver the presence and degree of swelling and inflammation of the optic nerve are in direct ratio to the rapidity of growth of the tumor rather than to the size of the mass. This is certainly true in cases of cerebellar tumor, in which optic neuritis of a high grade is usually an early symptom. The exact location of these tumors beneath the tentorium does not seem to be an important factor in the causation of optic neuritis. This symptom occurs as well in cases of tumor of the middle lobe of the cerebellum as in those of the cerebellar peduncles. This may be accounted for by the fact that a very small but active growth beneath the tense and unyielding tentorium soon causes, by pressure, active symptoms of various kinds.

The corpora quadrigemina, in which many of the optic neurons terminate, are very liable to suffer early in these cases by pressure or even by direct destructive processes. The optic neuritis in cases of brain tumor is usually double. This, however, is not an invariable rule. In some cases one nerve is involved before the other, or the grade of inflammation in one nerve may be greater than in the other, or the inflammation in one nerve may have partially subsided before its fellow is actively involved. According to Oliver the acute condition in one eye may subside sufficiently for a careless observer to assert that there has never been any inflammation in that nerve and that consequently the neuritis is unilateral. He refers to a case of his own which was under observation for a long time in which uniocular optic neuritis had been diagnosed and yet in which there were at times clear symptoms of a low grade of neuroretinitis in the fellow eye. The presence of unilateral optic neuritis in brain tumor is, however, of some diagnostic significance, although opinions differ as to the true value of this symptom. Many authors, as Jackson, Broadbent, Pagenstecher, have claimed that the inflammation is on the side opposite to the brain lesion. Others, however, deny this, and it is probably true, as Bramwell states, that the number of cases observed is too small to permit of a definite generalization as yet. Oliver, however, refers to two cases of double optic neuritis, both in children, in which the higher grade of neuroretinitis was on the side opposite to the intracranial lesions. In a recent case reported by Thomas and Keen[9] of a large brain tumor in the left frontal region the optic neuritis was greater on the side of the lesion, *i.e.*, the left side. In such a case it is possible that pressure was an important factor in the production of this symptom, as the lesion was a very large one and situated in a portion of the brain where direct pressure by a large mass downwards would be operative. In tumors, however, of the occipital lobe involving the optic centres in the cuneus, or

the optic radiations, it is probable that a higher grade of neuritis would be present in the eye of the opposite side, and this for the reason that the majority of optic fibres decussate in the chiasm. I do not wish, however, to make a positive assertion on this subject. In partial support, however, of such a view, Oliver refers to a case of sarcoma involving the pulvinar and contiguous portions of the internal capsule of the left side in which the right eye contained a large hemorrhage upon and around the optic disc. In brief it may be stated that the indications are that tumors of large bulk, in which the factor of pressure is a prominent one, may cause unilateral symptoms in the eye on the side of the tumor; but that, on the other hand, tumors of small bulk but of rapid growth in or contiguous to the optic fibres either in the optic tract or in the optic radiations within the brain, or near the optic centres in the cortex, may possibly cause unilateral symptoms in the eye of the opposite side. Such indications, however, are not sufficiently clear or numerous to admit of dogmatic statements.

The *course* of optic neuritis from brain tumor varies. The inflammation may subside without permanent impairment of the vision, but this of course is not a common result in such a steadily progressive lesion as brain tumor. Oliver attempts to give indications for the determination of the age or stage of progress of optic neuritis. In the very early stages a slight haziness of the nerve head with an indistinctness of the scleral ring and a slightly increased fulness of the veins may be the only signs present. In more advanced cases the disc will be swollen and projecting into the vitreous humor—the upper, inner, and lower portions, especially the papilla, being the most swollen. The outline of the disc will be obscured, the veins will be more swollen and tortuous and even lost to view at some points in or near their passage into the nerve, while the arteries will be greatly contracted. The appearance of striation caused by slight hemorrhages or by the prominence of smaller vessels becomes marked. From this acute stage the inflammation passes into what may be called a more chronic stage, and the swelling of the nerve head decreases, the outer or temporal border being first seen. The extravasations of blood will disappear. The vessels are no longer embedded in swollen retinal tissues and consequently can be traced farther and more distinctly upon the disc. They also become more nearly normal in appearance. Later the appearance of a postneuritic atrophy becomes still more marked; the outlines of the swollen disc (papilla) become more distinct; the contracting tissue squeezes the blood-vessels still more; the appearance of striation gradually disappears, and thus finally the nerve head or papilla presents the appearance of total atrophy.

The *prognosis* for the optic neuritis itself is not good as a rule in brain tumors. In those cases in which the initial swelling is very great and consequent loss of vision more pronounced, there may be improvement in sight after the diminution of this swelling takes place. The ultimate result to sight will of course depend upon the completeness of the inflammation and the number of fibres involved. As a rule there is slowly decreasing vision so that in many cases of brain tumor total blindness is the result, and in those cases in which this does not result it is probably because the fatal issue occurs before the degeneration of the optic neurons is complete. According to Oliver, in cases in which the neuritis has taken place without previous choking of the disc the vision gradually fails from the beginning. In view of the possible relief from brain tumors by surgical interference it is well to state that blindness in many of these cases is probably not the necessary result, even after a high grade of neuritis is reached, provided the neoplasm can be extirpated.

The *treatment* of optic neuritis in cases of brain tumor is of course quite impracticable. The only hope for its relief rests in the total extirpation of the neoplasm.

Abscess of the Brain.—Another cause of optic neuritis is abscess of the brain. This abscess is usually secondary to some suppurating process in other regions of the body. The most common cause is suppuration of the bones of the ear. Metastatic abscesses, however, in cases of general blood poisoning are occasionally seen. In rather rare cases the foci of suppuration are in the nasal or orbital bones.

As a symptom of abscess of the brain optic neuritis is of great importance, although it is not nearly so common as in cases of brain tumor. Its importance in cerebral abscess arises from the fact that it may in some cases confirm a diagnosis that might otherwise be obscure. In cases, for instance, of ear disease, in which the cerebral symptoms may be disguised, the presence of choked disc might furnish an important clew to the presence of an abscess in the brain. Oliver states that in the cases of children, if pus formation be rapid and extensive, pressure will soon ensue and give rise to optic neuritis. On the other hand, Keen[70] states that optic neuritis is not frequent in cerebral abscess, and again that the presence of choked disc does not seem to be pathognomonic, as this symptom is sometimes present and sometimes absent. Oliver claims that the optic neuritis of abscess of the brain is usually not so marked as is that of brain tumor, and that it is more likely to be unilateral and as a rule more decided in its onset. As is well known, abscesses of the brain have often a latent period, and it is during this period especially that the detection of optic neuritis might be of great importance. Gowers claims

that while inflammation of the optic nerve is less common in abscess than in tumors, it is probably more frequent than is suspected. He suggests that the ophthalmoscope would probably show that during the latent stage this important symptom frequently precedes the onset of the more acute symptoms. According to him it is more common in traumatic abscess than in other forms.

The statement of some writers that choked disc is found only in the very largest abscesses of the brain is not warranted by facts.

Optic neuritis has but little if any localizing value as a symptom of cerebral abscess. It will not permit of a differential diagnosis between a cerebellar and sphenotemporal abscess. Neither will it aid us in deciding upon which side of the brain the abscess is situated. In some cases it is reported as more marked upon the side opposite to that upon which the abscess is situated. It is even seen in cases in which the abscess is situated in the frontal lobe. In a recent case of abscess of the brain, Lannois and Jaboulay[11] observed hemianopsia on the side opposite the lesion. The abscess was situated in the left occipital lobe, and was associated with alexia, agraphia, word-blindness, and facial paralysis. It was caused by disease of the ear.

Basilar Meningitis.—Optic neuritis is much more common in this form of meningitis than in inflammation of the membranes of the vertex. In these cases it is probably caused by direct descent of the inflammation along the optic tract and nerve. Many authorities, as Bramwell, Hughlings Jackson, Gowers, and Oliver, state that the papillitis of meningitis is less intense than that of brain tumor. Gowers, for instance, says that usually the swollen papilla is paler than that which is found in other conditions. Oliver says that in the few cases observed by himself the venous engorgement was slight, while the disc was somewhat swollen and its edges hazy, but that the retinal hemorrhages ordinarily seen in other conditions were present in only one case. In tuberculous meningitis aggregations of tubercles may possibly be seen in the choroid. The changes in the nerve head and retina may be more pronounced, however, in tuberculous meningitis than in simple purulent meningitis, and this is probably due to the fact of the formation of tubercles in the choroid itself or to the masses of tubercles at the base of the brain acting like a tumor. The percentage of cases of optic neuritis in tuberculous meningitis has been asserted by Garlick, quoted by Oliver, to be as high as twenty-one out of twenty-six cases. It is possible that optic neuritis is more common at some stage of meningitis than the reports indicate, as many of these cases are probably not subjected to as careful and frequent ophthalmoscopic examination

as they might and should be. In the syphilitic form, for instance, optic neuritis is probably not uncommon. In fact, in hereditary syphilis this may be the cause of blindness.

Hutchinson reports a case of unilateral optic neuritis in a man of fifty who was the subject of extensive nerve syphilis. In such a case it is possible that the lesions began in a localized leptomeningitis at the base of the brain involving one optic nerve only. In such a case the presence of optic neuritis is of great diagnostic importance. In epidemic cerebrospinal meningitis optic neuritis is sometimes seen. It would probably be reported oftener in the grave forms of this disease were they not so rapidly fatal and so grave in character that an examination of the nerve head is not usually made because of the difficulties caused by the patient's condition. In these cases emboli and thrombi may be noted in the eye-ground. In spite of these grave conditions consecutive atrophy and permanent loss of vision are not common in cases which recover.

In hemorrhagic pachymeningitis there may be an inflammation of the optic nerves, but observations on this subject seem to be rare. Gowers calls attention to the importance of ophthalmoscopic changes in traumatic meningitis. In cases in which trauma leads to purulent meningitis about the base of the brain, optic neuritis may naturally be looked for; but there is a class of cases with fever, delirium, and even convulsions, not necessarily associated with basilar meningitis, in which optic neuritis may be seen. In cases in which the symptoms are only subjective the presence of optic neuritis, following trauma, might be of great diagnostic importance.

In sunstroke, or thermic fever, which is often associated with forms of meningitis, optic neuritis may appear. Hotz[13] claims that atrophy of the optic nerve is not uncommon in this country as a result of sunstroke. The arteries are narrowed as the result of inflammation. He observed these conditions not only after exposure to the sun, but after exposure to intense heat of other kinds. According to this author the inflammation is exudative in character and associated with choroiditis sufficient to cause detachment of the retina.

Insular sclerosis, according to Oliver, may cause optic neuritis if the patch be near the ocular globe. As a rule, this symptom is more marked on one side. According to Gowers, however, ophthalmoscopic signs are rare in this disease. He makes the statement that even in those cases in which patches of sclerosis occupy the optic nerve itself, the nerve fibres passing through are not destroyed, their axis cylinders persisting and retaining their function, only somewhat impaired. Such is not always the case, certainly, with these patches in other portions of the nervous system.

Diseases of the spinal cord may be associated with optic neuritis. Myelitis is one of these diseases. These cases are not very common, and when they occur the optic neuritis is probably caused by the same blood state, or poison, that causes the inflammation of the spinal cord. Katz relates a case in a woman of associated optic neuritis and acute myelitis. The symptoms were paralysis in the lower limbs and sensory impairment as high as the chest. The sphincters were affected. At the autopsy disseminated myelitis was found and atrophy of the optic nerves and the chiasm. Katz collected twenty cases. There were rapid loss of vision and swelling of the discs. Partial restoration of vision occurred in some, but complete blindness in others. The myelitis was either in the dorsal or lumbar region. Katz regards the affection as due to involvement of the sympathetic, as there is no direct anatomical connection—but this view is highly unsatisfactory. The best explanation is that a common cause (probably a toxæmia) exists for the inflammation in these dissociated regions of the central nervous system.

Syphilis.—When we consider the probability that syphilis is an active cause of myelitis or meningomyelitis; we can readily conceive that the optic nerves and tract may suffer from the presence of this poison in the blood. Syphilis, as we know, acts upon the nervous tissues, as a rule, through the vascular system. It excites an inflammatory process in the coats of the small blood-vessels. We have already seen that the development of the retina and optic fibres suggests a close analogy between them and the sensory fibres in the spinal cord. It is therefore unnecessary to suppose that there must be any direct anatomical connection between the cord and the optic fibres in order for them to suffer from the same poison in the blood. Syphilis seems to exercise a selective action, and thus probably tends to commit its ravages upon the optic sensory neurons at the same time that it invades the spinal cord. Locomotor ataxia, although not infrequently attended with changes in the optic nerve, rarely, if ever, presents acute or high-grade optic neuritis. Albutt described a stage of hyperæmia, or chronic optic neuritis, as preceding the optic atrophy so commonly seen in locomotor ataxia, but Gowers claims that this observation has not been confirmed by other observers. The atrophy of the optic nerve in locomotor ataxia will be described later.

Injuries to the spine are probably never attended with true optic neuritis. Attempts have been made in recent years to prove the contrary. This is no doubt due to the stimulus which this subject has received from litigation. Cases of railroad spine have been overstudied, symptoms have been exaggerated, and it is possible that some

observers have thought that they saw what they were looking for, when they noted minute and doubtful changes in the eye-ground of some of these litigants. Albutt's observations seem to prove, however, that ocular changes do occur in some cases of injuries to the spinal cord, especially at high levels. These changes are mostly congestion and undue redness of the disc, and they have been claimed by this observer to indicate a mild degree of neuritis. Certainly no very pronounced acute neuritis, however, is claimed, so far as I know, by any observer as occurring in the ordinary cases of railway spine. It is to be remembered, in this connection, that many of these cases have subjective hysterical symptoms, among which various limitations of the field of vision are not uncommon. This fact should put observers on their guard against misinterpreting subjective eye symptoms at least.

Bright's disease of the kidney is another cause of optic neuritis. The commonest form is the well-known albuminuric retinitis. This is seen especially in chronic cases; as in both interstitial nephritis and chronic parenchymatous nephritis. This form of retinitis, as its name implies, is usually associated with albumin in the urine, but its cause is evidently the same uræmic poisoning of the blood that accounts for the many other vascular changes seen in Bright's disease. Hence the amount of albumin in the blood, or even its presence there, is in no respect an indication or cause of this retinitis. Cases might occur in chronic interstitial nephritis in which little, if any, albumin is present. It is in such cases especially, in which the kidney disease has probably been insidious, with albumin not always present in the urine, that the importance of inspection of the eye-ground may be the greatest. A slight hypertrophy of the heart, with an accentuation of the second sound and a pulse of high tension, should indicate, therefore, the necessity for an ophthalmoscopic examination. In such cases the presence of the characteristic albuminuric retinitis may occasionally be even more confirmatory than an examination of the urine. According to Gowers, the objective symptoms of albuminuric retinitis are diffuse but slight opacity and swelling of the retina due to œdema, white patches due to the degenerative process, hemorrhages, inflammation of the nerve head, and later consecutive atrophy of the retina and nerve. These various signs vary in different cases, so that different types of albuminuric retinitis are recognized by ophthalmologists. In cases in which the white spots predominate the degenerative process is the most marked; in others the hemorrhages are the most conspicuous; in others again, the œdema and inflammation of the retina are most prominent, while in others still, inflammation of the nerve head may predominate. These various types,

however, are often associated or merge into each other, as, for instance, the degenerative and hemorrhagic conditions succeed the inflammatory state. The most characteristic type is that in which the white patches of degeneration and the retinal hemorrhages are the most conspicuous. These white spots of degeneration may appear near the disc, or at a distance from it. They tend to become larger and more irregular as the case advances. They are often especially conspicuous in or around the macula lutea, where they present a radiating form. In time they may form large areas.

The hemorrhages which are very constant in albuminuric retinitis may appear in association with the white spots. They also present a striated arrangement, caused by the course of the nerve fibres, among which they lie. They are sometimes seen in direct relation with the vessels; sometimes they are quite large.

In some cases of well-marked albuminuric retinitis the optic disc is not markedly affected; in others, however, the edge becomes obscured, while in cases in which the disc is inflamed it may be swollen, its edge completely obliterated, and its vessels concealed in the puffy tissue. In some few cases the swelling and inflammation of the optic disc are the most conspicuous symptoms, so that the condition simulates that seen in intracranial disease. Even in these cases, however, careful inspection will usually reveal small hemorrhages and signs of retinal degeneration. The subjective symptoms of albuminuric retinitis may be almost entirely absent. In course of time, however, affection of vision results, as, for instance, amblyopia and impairment of the color vision. Various limitations of the field of vision may be seen, as central or annular scotomata, caused by the degenerative patches in or near the macula lutea. Complete blindness is not common, although attacks of amaurosis sometimes occur even quite suddenly. In most cases death results from the advance of the kidney disease before the destruction of the optic fibres is complete. The course of albuminuric retinitis is, in fact, not always steadily progressive. Remissions occur, and Gowers states that retrogression may proceed until the changes noted disappear. The progress of the disease has been observed to be influenced even by treatment; active purgation may lessen it, while constipation, on the contrary, aggravates it. In the albuminuria of pregnancy, in which changes in the retina sometimes occur, complete cure may result after a successful delivery. As a rule, albuminuric retinitis furnishes ground for an unfavorable prognosis in chronic Bright's disease. According to Gowers such patients seldom live two years.

Local treatment for albuminuric retinitis is quite useless. The

only improvement possible is from general treatment for the state of the patient's blood, especially by purgation and diaphoresis.

Retinal hemorrhages are also seen not infrequently in *pernicious anæmia*. As a rule, however, in this disease, according to cases thus far reported, the signs of active inflammation of the nerve head and retina are not seen. The hemorrhages are probably due to the depraved condition of the blood, and possibly to some deterioration in the walls of the small blood-vessels. In our ignorance of the exact pathology of pernicious anæmia we cannot claim that the disease is due to any special blood poisoning, although the theory that it is thus caused is in accord with the clinical findings. In a case recently reported by the author,[13] these retinal hemorrhages were observed. This case occurred in a man who had been exposed to syphilis, alcohol, and malaria. On admission to the hospital he was markedly anæmic, the red blood cells being reduced ultimately to 800,000. The blood presented also many poikilocytes, with slight excess of leucocytes. At the autopsy degeneration of the posterior columns was found, and beginning slight degeneration of the lateral tract. An examination of the eyes was made by De Schweinitz,[14] who found the vessels of both retinæ diminished in size, and the retinæ themselves the seat of many hemorrhages. In view of the deterioration of the blood and blood-vessels of the retina which these findings suggest, it is perhaps allowable to suppose that cases may yet occur in which the optic nerve fibres may be found either inflamed or atrophied.

Disease of the Cranial Bones, especially when it involves the membranes, may cause optic neuritis. These cases, however, are properly to be classed with cases of meningitis. This disease is usually a caries associated with purulent meningitis, and the inflammation frequently extends to the optic tract or optic nerve by direct continuity.

Gowers refers to several cases of general hyperostosis of the bones of the skull in which optic neuritis or atrophy was observed. In some of these cases narrowing of the optic foramina had occurred, and this by causing pressure on the nerve trunk had probably excited an inflammation.

Orbital Inflammation.—A form of optic neuritis within the orbit is described by ophthalmologists. In this form the changes in the papilla are secondary to the inflammation of the nerve in the orbit, and are not always inflammatory in character, but may be simply atrophic, the result of the deeper seated inflammation. Such cases may be caused by an inflammation within or at the back of the orbit, such as the cellulitis caused by facial erysipelas. In these cases inflammation

may or may not be seen in the nerve head. Its presence there depends entirely upon whether the process extends thus far along the nerve trunk. In this connection it must be remembered that the cell bodies of the optic neurons are in the retina, that consequently the nutritive centres of these neurons are not impaired in such cases, and that, according to the Wallerian law, the descending degeneration would extend brainward, and not towards the retina. The fact is, however, that in some of these cases distinct evidences of atrophy are visible in the discs, and this may be due to the involvement of those optic fibres that grow from the brain into the retina. In some of these cases blindness occurs very early, and may not be associated with the visible signs of acute inflammation of the optic disc. In such cases exophthalmos, not necessarily of high degree, may occasionally be seen. Another form of orbital or retrobulbar optic neuritis is the chronic form caused by certain toxic agents, such as lead, alcohol, and nicotine. It is also seen sometimes in infectious diseases, such as rheumatism, diphtheria, and the various exanthemata. It has been described by De Schweinitz under the title, "Toxic Amblyopia." As in the acute form signs of active inflammation in the nerve head are often invisible. Occasionally, however, the disc may be slightly swollen and the color deepened, while a patch of consecutive atrophy appears later, especially in the lower and outer segment. The subjective symptoms of this condition are characteristic. These are diminution of vision and central scotoma, usually oval in shape, between the fixation point and the blind spot in which the perception of red and green especially is lost. The commonest causes for this retrobulbar optic neuritis are tobacco and lead. Other poisons, such as alcohol, quinine, opium, carbon bisulphide, and iodoform, and possibly also rheumatism, gout, and diabetes may cause it. The pathology is the same as that in peripheral neuritis. The process affects especially, for some unknown reason, the papillomacular fibres of the optic nerve.

In chronic lead poisoning, according to T. Oliver, atrophy of the optic nerve head is not infrequently observed. It is possible that most of these cases of atrophy result from a retrobulbar optic neuritis such as has already been described.

Changes somewhat similar to those seen in albuminuria and pernicious anæmia occur occasionally in *diabetes*. Hemorrhages and spots of degeneration may be observed in the retina. Ophthalmologists observe fine distinction between these spots and those of albuminuric retinitis. Nettleship[*] has depicted the changes due to retinitis in diabetes, and Gowers gives a minute description of the changes in the eye-ground due to diabetes. But as this subject be-

longs rather to ophthalmology than to neurology no attempt is made in the text at an elaborate description.

Infectious Diseases.—In typhoid fever instances have been reported of optic neuritis which, however, were probably due to associated meningitis. In typhus fever changes in the optic nerve are rare, and when they occur are probably the result of meningitis or the results of emboli or thrombi.

In relapsing fever Oliver says that choroiditis, cyclitis, and retinal and optic nerve complications may all appear. In scarlet fever, according to this author, double papilloretinitis has been observed without the presence of albumin in the urine. In such cases the changes in the optic nerve are probably due to a complicating meningitis. In the multiple neuritis following diphtheria grave optic nerve disease, while rare, has been seen. Oliver quotes a case in a boy aged twelve years who had double retinitis with partial degeneration of the optic nerve and who at the end of five years had marked reduction of normal vision in both eyes.

In measles optic nerve changes have also been observed. These are probably due to an inflammation of the nerve extending from a meningitis. In such cases the possibility of a localized tuberculous infection of the meninges must not be ignored. In whooping-cough changes in the eye-ground may be caused by hemorrhage from the rupture of a vessel during a paroxysm. Embolism may occur. Such complication is probably the most apt to occur in patients suffering with pneumonia and meningitis.

In small-pox, according to Oliver, deep lesions of the eye are infrequent. The case referred to by Gowers in which optic atrophy apparently dated from an attack of small-pox is open to some doubt, as the patient had symptoms of locomotor ataxia.

In malarial poisoning changes in the eye-ground are not unobserved. Oliver, however, thinks that affections of the optic nerve in these cases are rare. He says that the discs may become very pallid and white, but the retinal vessels are small, while limitations of the visual field are observed. In the malignant malaria of the tropics hemorrhagic retinitis with perineuritis may be observed, while fine striated hemorrhages may be seen in the retina. In some of these cases of retinal hemorrhages in malaria the optic nerves are not necessarily inflamed. During the paroxysm Ramorius, quoted by Gowers, observed that the nerve heads were pallid and the retinal vessels, both arteries and veins, were very small.

In yellow fever, according to Guitéras, emboli or thrombi may give rise to visual disturbance. In leprosy grave ocular changes may occur. The nerve substance may be infiltrated while the choroid and

even the retina may be involved. Pollock, quoted by Oliver, asserts, however, that no atrophic or pigment spots have ever been observed.

Optic Atrophy.

Atrophy of the optic nerve may be either primary or secondary. The latter is the result of a precedent inflammation, such as has already been described. In all cases of acute inflammation of the optic nerve a subsequent atrophy is the chief result to be feared. This of course indicates a destruction of the nerve fibres and if it is permanent results in impairment or loss of sight. The first or primary form of optic atrophy occurs without a precedent inflammation. In these cases, it is true, slight precedent congestion and redness may be observed, but in the main the process is a destructive atrophic one from the beginning. The question of its exact pathology cannot be discussed in detail here. It may be thought in some cases to be identical with the slow parenchymatous degeneration that characterizes so many of the systemic diseases of the central nervous system. This is the more probable since it is often associated with such diseases. We know, for instance, that locomotor ataxia is apparently due to a slowly degenerative process affecting especially the axis cylinder; that the signs of inflammation in the connective and vascular tissues are often not prominent. Optic atrophy is probably due to a similar process caused by the action of some toxic agent in the blood. If this view of the pathology of some forms of primary optic atrophy, especially that seen in locomotor ataxia, is correct, the process of degeneration is somewhat different from that following inflammation.

The appearances of optic atrophy are quite characteristic. These are especially increased pallor of the disc, increased distinctness in its outline or margin, and as a rule depression of the disc. In estimating variations in the color of the optic disc great care and expertness are essential on the part of the observer. The optic disc is normally of a delicate pink, not a dead white, and is in marked contrast with the redness of the surrounding eye-ground. Optic atrophy of course gradually affects this delicate color so that it is gradually lost and the disc is soon a dead white or gray color. In the beginning the pallor is most marked on the temporal side, where the fibres of the optic nerve are the least numerous. On the nasal side, in which the normal pink is most marked, the pallor becomes of course the more conspicuous by contrast with a normal disc. These fine shades of distinction can be determined satisfactorily only by an expert ophthalmoscopist. In advanced cases the whole disc is involved. Two forms of optic atrophy—the white and the gray—are described, but this distinction is of little clinical significance.

When the atrophy is complete the edge of the disc is even more distinct than in health; the outline is sharply defined; the sclerotic ring is more clear than normal but not always easily distinguished because it gradually merges into the pallor of the disc itself. The pigmentary ring at the edge of the disc may be unusually clear.

The disc itself, instead of being swollen as in optic neuritis, is usually depressed. This depression varies in different cases owing to variations in the amount of proliferation of the connective tissue. The peculiar mottling due to the lamina cribrosa may become unusually distinct. The blood-vessels in optic atrophy are not always changed in size. According to Gowers the vessels in gray atrophy undergo little or no change; in white atrophy the arteries are smaller, but the veins are not swollen or more tortuous; in fact the veins also may shrink.

The subjective symptoms of atrophy of the optic nerve consist of various forms of limitation of the field of vision passing into complete blindness. According to De Schweinitz these limitations consist of contraction, irregular angular limitations, quadrant-shaped defects, loss of one-half of the field, and abnormal blind spots or scotomata. In some of these cases of ataxia the red and green fields are markedly contracted.

The causes of optic atrophy are usually some blood states that cause degenerative diseases in other parts of the nervous system as well as the optic nerve. The most common of these undoubtedly is *locomotor ataxia*. The proportion of cases of tabes in which atrophy of the optic nerve occurs is perhaps not definitely ascertained. It is certainly very frequent; in fact, optic atrophy is seen more frequently in posterior sclerosis than in any other disease. Berger, quoted by De Schweinitz, gives forty-six per cent. as having been the proportion observed in his own cases. Gowers, however, gives a much lower percentage. Of seventy cases of tabes only nine presented atrophy. He quotes Voigt, who found it in nine out of fifty-two cases, and Erb, who found it in seven out of fifty-six cases. These results give only about fourteen per cent., which from my observation in the wards of the Philadelphia Hospital I should say was entirely too low. From the converse point of view, spinal symptoms appear in a very large proportion of cases of primary optic atrophy. We have the high authority of Charcot for the statement that nearly all cases of primary optic atrophy present spinal symptoms eventually. Gowers states that the proportion of these cases is about fifty per cent.

Atrophy of the optic nerves may appear early in locomotor ataxia, even before the onset of motor disorders. There is in fact a so-called sensory type of tabes in which fulgurant pains, in association with

atrophy of the optic nerves, are for a long time the only marked symptom. Brissaud [1a] refers to this type as being particularly liable to arthropathies. It may persist for many years before the onset of motor symptoms. In such cases the appearance of primary insidious atrophy should always excite the suspicion of beginning disease of the spinal cord or cerebrum. I now have under observation in the Philadelphia Hospital a man who has been blind for several years with complete primary optic atrophy, but who presents as yet very few symptoms of disease of the spinal cord.

Optic atrophy in locomotor ataxia usually appears in both eyes, although one eye may be affected before and to a greater degree for a time than the other. The atrophy is usually slowly but surely progressive, and sight in many of these cases is ultimately entirely lost. Loss of knee-jerks is an important confirmatory symptom in some of these sensory types of tabes. Instances are given of an extraordinary duration of this sensory type before the appearance of ataxic symptoms. Buzzard reported a case which existed for fifteen years. Gowers refers to one in which the atrophy lasted twenty years before the appearance of the ataxia. Although sight is lost in these cases the patients may walk with comparative steadiness, and this is in marked contrast with the ataxic gait of the motor type of cases in which, as is well known, the patient's ataxia is much increased on closure of the eyes.

The appearances of optic atrophy in locomotor ataxia are practically those that have already been described. The disc is gray in tone, the edge sharp, the sclerotic ring unusually clear, while the cancellations of the lamina cribrosa are often visible. The vessels as a rule are normal in size. The optic atrophy of locomotor ataxia frequently involves the whole course of these sensory neurons, hence it can be traced brainward through the chiasm as far as the external geniculate bodies. The change as found consists of an atrophy of the nerve fibre with some proliferation of the interstitial tissue. The weight of evidence seems to be in favor of the process being primarily parenchymatous, and its resemblance to that in the cord is usually considered by pathologists as striking. Gowers, however, seems inclined to dissent from this view. The one subjective symptom of optic atrophy in tabes is failure of vision. The last part of the field that disappears is usually about the fixation point. As the initial limitation of vision is in the periphery, the patient himself may for a long time be unaware that he is becoming blind. Color blindness, especially blindness for green and red, is an early symptom.

From what has already been said, it is evident that atrophy of the optic nerve in locomotor ataxia does not depend upon the atrophy of

the posterior columns of the spinal cord; that is to say, there is no direct anatomical connection between the two sets of fibres. In other words, the disease process in both is dependent upon a common cause; and this, according to the modern view, is probably some toxæmia, especially that caused by syphilis. The origin of the optic nerve, as already said, is in the cell bodies of the sensory neurons, located in the deep ganglionic layers of the retina; while the origins of the sensory neurons in the spinal cord are in the ganglia of the posterior roots. It seems probable therefore that in both cases the disease process begins independently in each set of neurons. This supposition is rendered more probable by the fact that the optic nerve, contrary to former teaching, is not a true peripheral organ, but is strictly analogous, from the embryological standpoint, as Monro has pointed out, with the sensory tracts in the spinal cord.

The prognosis of optic atrophy in locomotor ataxia is unfavorable. Recovery of sight is just as rare as recovery from the other symptoms of tabes. The progress, however, of the optic atrophy is sometimes exceedingly slow and many years may elapse before the patient is completely blind.

General paresis presents optic atrophy not unfrequently. The exact frequency of this symptom is a question of some doubt. The statement of Albutt that only 5 cases out of 53 presented normal papilla is probably misleading. A later observer, Uhthoff,[16] found atrophy of the disc in 8.04 per cent. of his cases. Of 167 cases recorded by Galezowsky and others (quoted by De Schweinitz) 7.02 per cent. presented optic atrophy. Other observers claim about the same figure, 5 and 6 per cent. being about a common average. Spitzka found atrophy in 3 cases of general paresis out of 39, while choked disc was seen in but 1 and pronounced hyperæmia in 4. According to this last observer the atrophy is found most commonly in those cases of general paresis that are associated with posterior sclerosis of the spinal cord. While this is undoubtedly true, it would be a mistake to suppose that this type alone of general paresis presents optic atrophy. I have seen it in a number of cases in the Philadelphia Hospital in which the symptoms of tabes were absent. This atrophy in general paresis is practically identical in appearance with that which has already been described in locomotor ataxia. It seems, however, according to Oliver, that the atrophy is especially marked on the temporal side—an observation that was verified by Spitzka. In addition to the optic atrophy, general paresis sometimes presents a slight discoloration of the disc and surrounding parts of the retina.

This degeneration of the optic nerve in general paresis, just as in

locomotor ataxia, is probably an expression of the general systemic affection rather than the result of the progress of the disease by direct continuity of the optic nerve with the cerebral centres. Clinically, just as in locomotor ataxia, it causes at first various limitations of the fields of vision, passing ultimately into total blindness. This result, however, may be long delayed.

Insular sclerosis not infrequently presents atrophy of the optic nerve. According to Uhthoff [4] who analyzed 100 cases—67 males, 33 females, the figures are as follows: 3 cases presented complete atrophy, 19 cases incomplete atrophy, and 18 cases atrophy of the temporal half of the disc, somewhat similar to that described in general paresis.

According to Uhthoff the changes in the optic nerve in multiple sclerosis have special characteristics that distinguish them from other forms of atrophy. They stand midway between tabetic atrophy on the one hand and postneuritic atrophy on the other. The changes appear to resemble those of interstitial neuritis marked by proliferation and growth of nuclei. Atrophy of the nerve substance follows as a secondary result. The medullary sheaths are destroyed rapidly, the axis cylinders being preserved permanently in many places. The blood-vessels undergo changes in their walls with proliferated tissue around them. Descending degeneration of fibres is sometimes absent. The papilla may even maintain its normal appearance although extensive retrobulbar atrophy has occurred. Uhthoff speaks evidently of descending degeneration passing from the brain to the eye. We know now, however, that the trophic centres of many of the optic fibres are in the cell bodies of the neurons of the deep ganglionic layer of the retina. It is evident, in fact, from Uhthoff's description that the most intense change often occurs in the retrobulbar portion of the optic nerve and that the papilla and retina show comparatively few changes. This evidently indicates that the trophic centres of these optic neurons are not primarily involved.

Uhthoff gives the following table of the sex and age of his one hundred patients with multiple sclerosis:

Age.	Males.	Females.	Age.	Males.	Females.
1 to 10	4	2	40 to 50	16	8
10 to 20	2	4	50 to 60	4	8
20 to 30	18	9	60 to 70	1	0
30 to 40	22	12			

Optic neuritis was present in 5 cases; it was monocular in 3 and binocular in 2. Vision was affected in 4 out of the 5 cases. The appearances were normal in 48 cases. In 5 of these, however, there was disturbance of vision, and in 1 of the latter cases changes in the op-

tic nerve were discovered upon microscopic examination after death. Among changes noted in the fields were central scotoma with intact periphery, central scotoma with peripheral contraction, irregular peripheral contraction with relatively good central vision, and in 1 case regular concentric contraction of the field. The course of the amblyopia was traced in 22 of these cases. In one-half it came on rapidly, in the others gradually. In some cases improvement of vision occurred and in 2 cases recovery of normal vision was noted. In 4 cases the amblyopia was an initial symptom preceding all other symptoms of the multiple sclerosis by as much of an interval as from two to three years. The first symptoms were those of retrobulbar neuritis.

Uhthoff gives statistics also of paralysis of the ocular muscles met with in his one hundred cases, as follows:

Paralysis of the ocular movements present in 17 cases, viz., paresis of the sixth nerve, 6 cases (binocular in 2, monocular in 4). Paresis of the third nerve, 3 cases (monocular and partial in each instance, in 1 case the levator of the lid and superior rectus being affected, in 1 the internal rectus and in 1 the superior rectus). Paresis of associated movements, 2 cases (of lateral movements in both directions in 1, towards the left in 1, upwards in the third). Paresis of convergence in 3 cases. Pronounced ophthalmoplegia externa in 2 cases. Nystagmus was met with in 58 of the cases. Involvement of the pupils was rare, being found only in 11 of the 100 cases.

It thus appears that insular sclerosis ranks next to locomotor ataxia in the frequency with which it presents optic atrophy. De Schweinitz[14] regards the amblyopia of multiple sclerosis as most resembling non-toxic retrobulbar neuritis. According to Gowers this amblyopia, however, rarely goes on to complete loss of sight. He even claims that it may be unattended by the ophthalmoscopic signs of atrophy. In some cases the optic nerves, he says, are occupied by patches of sclerosis just as in the central nervous system. The nerve fibre, he thinks, may pass through these patches intact.

In Friedreich's ataxia atrophy of the optic disc has been observed by Power and Seguin, quoted by Griffith.[15] In this latter author's elaborate study these two cases alone out of one hundred and forty-three cases presented optic atrophy. To be sure, he says that ophthalmoscopic examinations had been made in only about thirty-eight of these cases. In none of them was any change of importance noted excepting in Power's case, in which the discs were rather white, and in one of Seguin's, in which there was partial atrophy of both optic nerves. Oliver found narrowed field in one of Sinkler's cases and this he attributed to changes similar to those seen in tabes. De

Schweinitz states that he has made similar observations. On the whole, according to Griffith's table, the study of the eye-ground in this disease has been too much neglected, and it is possible the changes in the optic nerve may be found yet to be more common than is generally supposed. In a well-marked case of Friedreich's ataxia at present under my observation in the Philadelphia Hospital the visual fields are slightly contracted.

In amyotrophic lateral sclerosis atrophy of the optic disc is not commonly observed. Isolated cases are on record, however, as that of Petelsohn, quoted by De Schweinitz.

In lateral sclerosis it is also practically unknown. I do not recall an instance of it in my observations at the Philadelphia Hospital. Gowers states that he has seen it once.

In injuries of the spinal cord optic atrophy is not observed. The opinion that it sometimes occurs seems to be founded originally on the assertion of Albutt, but this has not been generally confirmed.

According to Oliver the various infectious diseases sometimes show changes in the optic disc.

Diseases of the Third Nerve.

The third or oculo motor nerve supplies most of the muscles of the orbit as well as the iris and ciliary muscle. It arises from a series of nuclei containing large ganglionic motor cells just beneath the aqueduct of Sylvius. The most anterior of these ganglionic cells lie in the walls of the third ventricle. From this point the nuclei extend backwards almost the whole length of the aqueduct. The roots of the nerve emerging from the nuclei pass ventrad through the tegmentum and the crusta to the inner side of the cerebral crura. The nerve emerges on the inner surface of the crus cerebri. As it emerges from the brain it receives a sheath of the pia mater and of the arachnoid. After piercing the dura mater it passes along the wall of the cavernous sinus, being situated above the other orbital nerves. It receives in its course a few fibres from the sympathetic. It enters the orbit by two branches through the sphenoidal fissure. It is here placed below the fourth nerve close to the frontal and lacrymal branches of the ophthalmic. Of its two divisions the superior, or smaller, supplies the superior rectus and the levator palpebræ muscles. The larger, or inferior branch, subdivides into three branches. One passes beneath the optic nerve and supplies the internal rectus. The second passes to the inferior rectus. The third passes to the inferior oblique. Fibres are also supplied by the nerve to the iris and ciliary muscle.

The arrangement of the nuclei of the third nerve in the midbrain and the relations of these nuclei to the nuclei of the fourth and sixth nerves are of great clinical interest. These nuclei, as pointed out by Edinger, probably consist of a complex of smaller nuclei somewhat separated from each other. He thinks that in human beings a distinct demarcation can be made out. The most anterior is a small nucleus lying even partly in the wall of the third ventricle. Farther back the series of nuclei of the third nerve evidently tends to become separated into groups. One of these groups gives origin to fibres which decussate within the structure of the midbrain with those of the opposite side. The fibres from the other nuclei do not decussate but pass out by the nerve trunk on the same side. Edinger thinks that it has been satisfactorily determined that the nuclei of the third nerve supply the individual muscles of the orbit in the following order from before backwards:

Sphincter iridis, Levator palpebræ, Rectus superior, Obliquus inferior,	Musculus ciliaris, Rectus internus, Rectus inferior,	Middle line.

According to this arrangement the nerves to the internal muscles, the iris, and ciliary muscles, arise from the foremost of these nuclei. The decussating fibres probably pass to the internal rectus. The relation of these nuclei with the optic nerves raises questions of much clinical importance. That there are such connections is indubitable, because the eye reflexes and the various associated movements of the eye must depend upon such anatomical connections. Edinger says that such an anatomical basis has not yet been demonstrated, although numerous fibres exist which might form the communication. That the various muscles of the orbit and of the interior of the eye have their individual nuclei is made evident from the clinical fact that degenerative diseases sometimes occur in which some, but not all, of these muscles are affected or in which some are affected before others. The paralysis caused by disease of the nuclei of the third nerve and of the nuclei of the associated fourth and sixth nerves is called ophthalmoplegia. It may be partial or complete and its various forms will be described later, after the description of the individual diseases of the third, fourth, and sixth nerves.

The third nerve may be affected by inflammation, by pressure, or by trauma.

Inflammation of the third nerve is a very common symptom of syphilitic meningitis at the base of the brain. Ricord has said that "syphilis places its sign manual on the third nerve." In some cases the nerve is paralyzed without evidence of involvement of other struc-

tures, even the meninges. In such a case it is possible that the inflammation may be entirely limited to the sheath of the nerve or its trunk. Diphtheria also may cause inflammation of the third nerve or some of its branches. In locomotor ataxia paralysis of individual muscles supplied by the third nerve is sometimes seen, thus the levatores palpebrarum may be affected or paralyzed. The external

FIG. 16.—Complete Paralysis of the Right Third Nerve in a Case of Syphilis of the Brain. (From a photograph of a patient in the Philadelphia Hospital.)

rectus and superior rectus are also not uncommonly involved. In some cases all the muscles supplied by the third nerve are involved in tabes. Both eyes or only one may be affected, the same muscles not being necessarily involved, or involved to the same extent in both eyes. In tabes, however, it is still a question whether these ocular palsies are due to involvement of the nerve trunk or to that of the nuclei. The degeneration of tabes is so nearly confined in all parts of the nervous system to the sensory fibres that it may be a question whether in this instance the third nerve forms a true exception to the rule.

Alcoholic neuritis does not involve the third nerve or its branches. Cases of inflammation of this nerve occasionally occur after exposure

to cold. In such cases Gowers is inclined to think that the cause of the inflammation may be a rheumatic poison, but in all such cases the action of a possible syphilitic infection must be rigidly excluded. Charcot[14] has described a rare form of paralysis of the ocular muscles associated with migraine. These cases, like the migraine itself, are recurrent, and after enduring for a period of days or weeks recover. The paralysis falls not only upon the branches which supply the exterior muscles, but also upon those which supply the interior muscles, *i.e.*, ciliary muscles and iris. It is consequently a total ophthalmoplegia. Such cases have been reported by Manz, Senator, and others. Charcot calls this affection "migraine ophthalmoplégique." The pathology of the disease is uncertain, but an observation of Gubler[15] upon a patient who had died seems to prove that the oculomotor nerve may be enveloped in an inflammatory exudation. Charcot seems inclined to believe that the repeated accesses of the paralysis due to the underlying cause which produces the migraine predispose the nerve to an inflammatory or exudative process, but this theory is as yet only tentative. Vasomotor disturbances are supposed by some to be the primary underlying cause of the affection, but this explanation fails to explain. The fact, however, that the occurrence is paroxysmal, and that the paralysis of the nerve gradually passes away seems to prove conclusively that the cause itself is not permanent. That the affection may be nuclear in origin is not improbable. Against this view, however, is the fact that usually the third nerve alone is involved and very frequently on one side only. For the present the pathology of this rare affection must be considered obscure. The paralysis may last for only a few days, or in severe cases for as long as from three to eight weeks. When the paroxysms occur at long intervals the paralysis as a rule lasts for a longer time than when they occur frequently.

In tuberculous meningitis the third nerves may be involved in the exudative process at the base of the brain.

Pressure upon or involvement of the third nerve may be caused by a tumor, or an aneurysm at the base of the brain. Tumors or hemorrhage in the crura cerebri very frequently involve the third nerve. Thus in a case reported by the author[16] a tumor of the midbrain involved the motor fibres passing downwards from the brain, and the fibres of the oculomotor nerve on the same side. Such a lesion causes the well-marked cross paralysis (or syndrome of Weber) in which a hemiplegia of the opposite side coexists with paralysis of the oculomotor nerve on the same side as the lesion. Tumors at the base of the brain may involve one nerve, or both, according to their location.

Various forms of trauma may cause paralysis of the third nerve or some of its branches. The most common of these are injuries within the orbit, especially gunshot and pistol-shot wounds.

The *symptoms* of affections of the third nerve are highly characteristic. When the trunk of the nerve is involved they are as follows: The internal rectus muscle is paralyzed, consequently the eyeball is turned far outwards under the influence of the external rectus, which is supplied by the sixth nerve. The upper eyelid droops so as to completely obscure the eyeball. The pupil is widely dilated, because its constrictor fibres alone are paralyzed. The eyeball is almost immobile because it cannot be rolled upwards or downwards by the action of either the superior or inferior rectus muscles. If the patient attempts to raise the upper eyelid the occipito-frontalis muscle is strongly contracted. Because of the paralysis of the constrictor fibres of the iris the pupil does not react to light nor upon accommodation.

If the branch of the third nerve that supplies the internal rectus alone is paralyzed the symptoms are as follows: The eye cannot be turned inward; in other words, the patient has divergent strabismus. He also has diplopia or double vision. If the muscle affected is on the right side the image exists in the left half of the motor field, and *vice versa*.

If the branch to the superior rectus is paralyzed the upward movement of the eye is almost but not quite abolished. The characteristic feature of this movement is that the eyeball is rotated by the inferior oblique muscle, which tends also to rotate it upwards. Diplopia is also seen. The images are placed one above the other, and the false one is the higher and is slightly inclined. If the branch to the inferior rectus is involved the downward movement of the eyeball is affected, but not absolutely so, as the superior oblique muscle tends to draw the eyeball downwards as well as inwards, and thus supplements to some extent the action of the affected rectus muscle. Diplopia is present in the lower part of the field of vision. The false image is below the true image. On attempting to look downwards the upper lid does not descend as it does when the inferior rectus is normal.

When the branch that supplies the inferior oblique muscle is affected the movement inwards and upwards of the eyeball is impaired. Diplopia exists especially in the upper part of the field.

If the branch to the levator palpebræ muscle is involved the eyelid droops so that it almost or quite covers the eyeball. The eyebrow is usually somewhat elevated from overaction of the frontalis muscle, a highly characteristic sign. This is probably due to the fact that

normally the frontalis muscle acts with the elevator of the upper lid, and that consequently when the upper lid is paralyzed the attempts of the patient to innervate it cause an increased action of the unparalyzed frontalis muscle.

A case of third-nerve paralysis occurring in the author's clinic in the Philadelphia Hospital illustrated some characteristic symptoms. The patient was a young man with a history of syphilis in whom third-nerve paralysis had come on with some headache, but no other well-marked symptoms. In his case the internal rectus muscle was entirely paralyzed so that the eye was turned widely outwards. Upward and downward movements were also abolished except a slight downward and inward movement caused by the action of the superior oblique. The pupil was widely dilated and immobile to light and on accommodation. The ptosis or drooping of the upper lid, however, was not complete, although the patient had very little apparent power remaining in the muscle of that lid. This patient made a satisfactory recovery under antisyphilitic treatment. During his stay in the hospital he did not develop any other complications on the part of the nervous system. His case was quite a characteristic example of syphilitic infection of the third nerve. (Fig. 17.)

FIG. 17.—Paralysis of the Left Third Nerve, with Partial Drooping of the Upper Lid. (Philadelphia Hospital.)

The internal muscles of the eye—*i.e.*, the iris and the ciliary muscle—being also supplied by the third nerve, are paralyzed when the trunk of that nerve or the branch through which their fibres

run is affected. As the fibres of the third nerve supply only the constrictor muscles of the iris this action alone is impaired. This paralysis is called iridoplegia. As the dilator fibres, however, which are under the control of the sympathetic nerves are not paralyzed the pupil is widely dilated, so that it cannot be said in the third-nerve palsy that the iridoplegia is complete, as only one set of fibres—*i.e.*, the constrictor fibres—is impaired. This permits such complete overaction of the dilator fibres that the pupil is stretched widely open, these dilator fibres acting to their utmost capacity. Hence the pupil is practically immobile, being unable to contract because the constrictor fibres are paralyzed, and being unable to dilate further because it is already fully dilated. The reflexes of the iris are consequently entirely abolished; thus the iris will not react to light by contracting the pupil, and, being already fully dilated, cannot react farther to irritation of the sympathetic fibres on the side of the neck and face to dilate the pupil. Its power to contract, and consequently to vary the size of the pupillary orifice on accommodation, is also entirely lost. As a consequence of this latter loss the patient's vision for near objects in the affected eye is blurred. When the patient attempts to look at a near object the pupil does not contract.

As the ciliary body is usually paralyzed at the same time as the iris in third-nerve paralysis, the power of accommodation itself is lost, hence vision for near objects, as already said, is blurred. This paralysis is called cycloplegia. The patient cannot read with the affected eye nor distinguish small objects close at hand. Distant vision, however, may remain good. In myopia, however, and presbyopia, the absence of power of accommodation is not so notable. Cycloplegia or paralysis of the ciliary body is a common symptom in diphtheritic paralysis, in which case it is sometimes associated with paralysis of some other branches of the third nerve. The seat of the paralysis, however, in diphtheria may possibly be in the nuclei of this nerve in the midbrain, rather than in the trunk of the nerve itself.

In some cases of iridoplegia due to third-nerve paralysis the pupil is not so widely dilated but that a still further dilatation may be obtained by the so-called skin reflex, *i.e.*, by irritating the sympathetic fibres on the side of the neck and face. As this reflex does not travel through the third-nerve fibres, it is of course not really abolished in any instance of paralysis of this nerve, but only rather disguised by the excessive dilatation of the pupil.

The subjective symptom of paralysis of the third nerve is especially diplopia or double vision. The exact physiology or psychology of this symptom is not easily explained. It depends of

course upon the fact that the eyes no longer act harmoniously, the axes of the eyes either diverging or crossing each other in abnormal ways. Gowers[4] attempts to explain this symptom from the psychological standpoint. He seems to think that it depends upon an error of judgment depending upon the fact that we judge of the relation of external objects to our own body by the position of the eyeball as indicated to us by the amount of innervation we give to the ocular muscles. In other words, we depend for this knowledge upon a muscular sense. Hence when one of the ocular muscles is weakened the sense of increased innervation needed to move this muscle is falsely interpreted, and the mind makes an erroneous interpretation of the relative position of the two images. This explanation seems somewhat obscure and unsatisfactory, although it is probably impossible to give one that will adequately explain the facts. From a clinical standpoint it is probably sufficient for us to know the fact without attempting to theorize about it. The "true image" is the one formed by the unaffected eye; the "false image" being the one formed by the affected eye.

The third nerve being entirely motor there is no sensation of pain due to a pure oculomotor palsy. If pain occurs in any of these cases it is due to the underlying cause, such as the meningitis or pressure by a tumor that involves the nerve. In some cases, however, a distinct sensation of giddiness or vertigo results.

The *course* and *prognosis* of oculomotor palsy depend of course upon the lesion that produces it. If it is caused by a destructive lesion, such as degeneration of the nuclei, or a brain tumor, or an extensive incurable meningitis, or an incurable trauma, it will most probably be either steadily progressive or permanent. In cases in which it is caused by the poison of syphilis, provided concurrent symptoms of a more extensive lesion are absent, the prognosis is often favorable. Active antisyphilitic treatment may relieve and even permanently cure such cases. In the rare instances to which Gowers refers, in which the affection seems to be due to exposure to cold, the prognosis is also favorable.

The *treatment* of oculomotor palsy depends of course upon the underlying cause. In the syphilitic cases, as already said, an active specific treatment is indicated. This is best met with inunctions of mercury or the hyperdermic use of some mercurial preparation, associated with large doses of the iodides. In the simple rheumatic cases the salicylates may be used, and the use of a mild galvanic current is probably warranted. Extreme caution, however, is necessary in the use of electricity about the eye; only very mild currents should be employed. In the grave cases caused by destructive

lesions, such as tumors, meningitis, and degenerative processes in the nuclei, little can be done for the relief of this paralysis.

Diseases of the Fourth Nerve.

The fourth or pathetic or trochlear nerve is the smallest of the twelve cranial nerves. It supplies but one muscle, the superior oblique. It arises from a nucleus somewhat posterior to the nuclei of the oculomotor nerve. This nucleus is situated at the level of the depression midway between the anterior and posterior corpora quadrigemina (Bruce[88]). It is separated by a very slight interval from the posterior end of the third nucleus. The root of the nerve passes backwards and then inwards and decussates in the substance of the valve of Vieussens or anterior medullary velum with its fellow of the opposite side. This nucleus is connected with the posterior longitudinal fasciculus, a band of fibres that evidently connects the various nuclei of the orbital nerves. According to Bruce this decussation of the root of the fourth nerve, which is the only instance of such a decussation of a cranial nerve, has been denied by a number of observers. Von Gudden, however, showed that extirpation of one trochlear nerve in new-born kittens was followed by complete atrophy of the opposite fourth nerve (Bruce). The nerve emerges from the posterior medullary velum behind the corpora quadrigemina and passes around the outer side of the cerebral peduncle. It passes forward to the outer wall of the cavernous sinus near and below the third nerve, and enters the orbit through the sphenoidal fissure. It enters the superior oblique muscle on its orbital surface. In its course the fourth nerve receives filaments from the sympathetic. The fourth nerve is the uppermost nerve in the orbit.

As the fourth nerve is the smallest and simplest in its origin and distribution of all the nerves of the cranium the symptoms of its diseases are few and easily understood. The nerve supplies only the superior oblique muscle. The action of this muscle is to rotate the eyeball downwards and inwards. It also rotates the eyeball on its antero-posterior axis, consequently a paralysis of this muscle interferes with the movement and permits the ball to be rolled too far upwards and inwards by the action of the unantagonized inferior oblique muscle. The result of this is to cause a convergent strabismus when the patient attempts to look down. This causes diplopia only during the same movement of the eye, a condition which may be especially troublesome and dangerous when the patient goes downstairs (Herter).

Isolated disease of the fourth nerve is extremely rare. I know of

no instance of it. It is conceivable that the fourth nerve might be injured within the orbit without injury to other nerves, but cases of this kind must be exceedingly rare. Brissaud presents a diagram of a conjoint lesion of the sixth and fourth nerves due to a pistol-shot wound. This nerve is never affected alone by syphilis nor by meningitis at the base of the brain. Tumors of the orbit or of the base of the brain could hardly be so small and so located as to affect this nerve only. The only probable lesion that could affect the fourth nerve alone would be degeneration of its nucleus in the midbrain.

The pathology of all such probable lesions has already been indicated in the description of the third nerve.

No special *treatment* need be described for an affection so rare and so inaccessible as paralysis of the fourth nerve. In cases in which this nerve is paralyzed along with the third and sixth nerves, due to degeneration of the nuclei, little can be accomplished. In cases in which it is involved along with other nerves in syphilitic meningitis at the base of the brain the treatment of course would be by mercury and the iodides.

Diseases of the Sixth Nerve.

This nerve, also called the abducens, arises from a nucleus somewhat remote from the nuclei of the other ocular nerves. This nucleus is situated in the tegmentum of the pons just beneath the under surface of the fourth ventricle. It is surrounded or embraced by the knee of the seventh nerve. The root of the sixth nerve arising from this nucleus passes forwards through the tegmentum of the pons and emerges on its under surface, just at the junction of the pons with the medulla oblongata. It passes forwards near the other orbital nerves in the wall of the cavernous sinus and enters the orbit through the sphenoidal fissure. Passing between the two heads of the external rectus muscle it is distributed entirely to this muscle.

The action of this nerve, because of its limited distribution, is very simple. As it supplies the external rectus muscle alone it presides only over the outward movement of the eyeball, and therefore its paralysis allows the eyeball, under the influence of the unopposed internal rectus, to be turned far inwards. The patient consequently has a highly characteristic internal strabismus. This nerve is not infrequently affected alone. Its liability to damage is increased by its long and exposed course. It may be affected in cases of syphilitic meningitis without involvement of any other cranial nerve. It may also be involved in a circumscribed lesion of the pons. In this way it is sometimes paralyzed in association with the seventh nerve. It

might possibly also be involved in traumata of the orbit, although such cases are rare. In some comparatively rare instances paralysis of the sixth nerve is associated with polyuria or diabetes insipidus. This is possibly due to its nucleus in the pons being contiguous to the diabetic centre in the floor of the fourth ventricle. This curious association is seen in a case at present under my care in the Phila-

FIG. 18.—Paralysis of the Right Sixth Nerve. (Upson.)

delphia Hospital. The patient, a young man about twenty-four years of age, has had total paralysis of the sixth nerve accompanied with great polyuria. The hitsory of his case is as follows:

W. C —— 21 years, white, male, had a history of syphilis. Before admission to the hospital he had shown mental changes of slight degree, such as inattention to work, etc. He was brought to the hospital in a semi-conscious state. There was no paralysis of motion or sensation. His symptoms since his admission have been as follows: Complete paralysis of the right sixth nerve; slight drooping of the right side of the face; no involvement of the fifth or of other cranial nerves, except the sixth, and possibly the eighth as there is some dulness of hearing; slight staggering gait, with a tendency to go to the left; marked polyuria. The examination of the eyes revealed no changes of importance in the eye-grounds. Later the patient had several apoplectiform crises, with slight clonic spasms in the right upper and lower extremities. About three months after admission the paralysis of the sixth nerve entirely dis-

appeared, and was replaced still later by a paralysis of the third nerve. The upper eyelid drooped, the eyeball was turned outwards, and the pupil was dilated. There was diplopia. The patient now presented anosmia. The polyuria, a daily record of which was kept for many weeks, was marked by a daily flow of from fifty to as high as one hundred and thirty ounces. There was no sugar nor albumin

FIG. 19.—Paralysis of the Right Sixth Nerve in a Case of Syphilis of the Brain. Development of the right mammary gland. (Philadelphia Hospital.)

in the urine. Progressive emaciation set in until the patient had the appearance of advanced muscular atrophy. As anomalies in this patient may be noted a very high palatal arch, deficient hair on the pubes, and a remarkable development of the right mammary gland. (See Fig. 19.)

Disorders of the orbital muscles and of sight have often been observed in diabetes insipidus. Spanboch and Steinhaus record a case in a woman of bitemporal hemianopsia and polyuria. The authors

suppose that there were two foci of disease—one in the chiasm and one near the nucleus of the vagus. It is more probable that there was a diffused basilar syphilitic meningitis, involving the chiasm and the vessels running up into the pons.

The pathology of affections of the sixth nerve has already been indicated in what has preceded.

FIG. 20.—Paralysis of the Right Sixth Nerve in an Idiotic Child. (Elwyn Institute.)

In syphilitic cases the *treatment* of course is by specific medication. In other instances treatment should be directed to the underlying cause. In destructive lesions of the pons, such as hemorrhage, tumor, or degeneration of the nuclei, practically nothing can be done. In cases of traumata about the orbit any possible treatment would be surgical.

Combined Palsies of the Ocular Muscles.

The ocular muscles may be paralyzed in various combinations. Thus, as already said, the external rectus muscle may be paralyzed in association with the muscles supplied by the seventh nerve. Such a paralysis is probably due to a focal lesion in the pons in, or in the neighborhood of the nucleus of the sixth nerve and involving the fibres of the seventh nerve which course around it. The true combined palsies of the ocular muscles are very various. Hutchinson[1] in 1879 called attention to a form of combined paralysis which he called ophthalmoplegia or symmetrical paralysis of the ocular muscles. He borrowed this term, however, from von Graefe and Eulenburg, who had described the condition before him. By this term Hutchinson meant to describe especially a condition in which all the extrinsic muscles of the eye are paralyzed, but in which as a rule the internal muscles, *i.e.*, the iris and ciliary muscle, escape. According to Hutchinson its best marked examples occur in adults who have had syphilis many years before, but it has also been observed in patients with inherited syphilis and in some few instances it has occurred without a known cause. In this form of paralysis, as a rule, all the external muscles of both eyeballs are more or less involved, and in some cases the paralysis is progressive and becomes complete, so that the eyes are motionless in their sockets. Under these circumstances the patient can change the direction of his gaze only by moving this head. In most instances, however, according to Hutchinson, the paralysis does not become absolute and it rarely affects all the muscles in the same degree. One or two may even escape entirely. There is always some ptosis, but this varies in different cases and is rarely complete. This ophthalmoplegia externa may, however, in some cases be associated with ophthalmoplegia interna, *i.e.*, with paralysis of the iris and ciliary muscle. In some cases, according to Hutchinson, atrophy of the optic nerve with blindness results.

In recent years authors have attempted to classify the various forms of ophthalmoplegia according to the number of muscles involved and the completeness of the paralysis. Brissaud proposes the following classification: Ophthalmoplegia may be first total and complete; second, total and incomplete; third, partial and complete; fourth, partial and incomplete.

Ophthalmoplegia is total and complete when all the ocular muscles are paralyzed and when this paralysis is complete or absolute in degree. It is total and incomplete when all the muscles are

paralyzed, but when the paralysis is incomplete, *i.e.*, when the functions of the paralyzed muscles are not absolutely abolished. Again, ophthalmoplegia may be partial and complete. That is to say, only a portion of the musculature of the eye is involved, but this portion is completely paralyzed. Finally, ophthalmoplegia may be partial and incomplete. In this case one or several muscles (but not all) are paralyzed, but incompletely so—*i.e.*, their function is not completely abolished.

This classification covers all possible forms of ophthalmoplegia, especially if it is designated in addition whether the paralysis is bilateral or unilateral. Thus, for example, a complete paralysis of all the muscles (both internal and external) of both eyes would be designated a bilateral total and complete ophthalmoplegia. The distinction, observed by Hutchinson, between ophthalmoplegia externa and interna, is not so important, because instances are extremely rare, in which this distinction is observed by the process of disease. Hutchinson, however, insists in his paper that such distinctions do occur sometimes.

Bilateral total and complete ophthalmoplegia is undoubtedly always due to a destructive process in the nuclei of the third, fourth, and sixth nerves. It may also be caused by the compression of a brain tumor upon the roof of the midbrain.

As an example of ophthalmoplegia, partial and complete, we might suppose a paralysis of the third and fourth nerves on both sides caused by the pressure of a tumor in the interpeduncular space; in such an instance the sixth nerve might escape, as its point of exit from the pons is some distance away. The paralysis in such a case would fall upon all the muscles supplied by the third and fourth nerves; consequently, both eyes being left to the influence of the sixth nerve, would be turned far outwards.

A unilateral total and complete ophthalmoplegia would probably indicate a lesion in the orbit involving the third, fourth, and sixth nerves. Such a paralysis, to be sure, might be conceived as caused by a destructive process in the nuclei of these nerves in the midbrain and pons on one side, but such extensive unilateral nuclear disease would not probably occur.

An ophthalmoplegia, total but incomplete and bilateral, would indicate a lesion of the nuclei of the third, fourth, and sixth nerves in the midbrain and pons that had not yet at the moment of observation become completely destructive.

A bilateral ophthalmoplegia, partial and complete, would also indicate a nuclear degenerative or destructive process which at the moment of observation had invaded and completely destroyed only

some of the nuclei and had as yet spared the others. It would be the same if such an ophthalmoplegia were incomplete.

Finally, a partial unilateral ophthalmoplegia, either complete or incomplete, would indicate a lesion not in the nuclei but somewhere in the course of the nerves involved, *i.e.*, upon one or more branches of the ocular nerves.

The complete bilateral ophthalmoplegia interna—*i.e.*, paralysis of the iris and ciliary muscle without involvement of any of the extrinsic muscles of the eye—would indicate undoubtedly a lesion of the first and second pair of nuclei of the third nerve group, *i.e.*, those lying near the wall of the third ventricle.

The complete bilateral ophthalmoplegia externa of Hutchinson undoubtedly constitutes a type and is always due to a lesion of the nuclei of the third, fourth, and sixth nerves in the midbrain and pons. It is a matter of comparatively minor importance, from a diagnostic standpoint, whether or no this type ultimately is associated with paralysis of the interior muscles of the eyes. In such a case the indication simply is that the nuclei of these interior muscles, lying most anterior near the wall of the third ventricle, have become involved in the destructive process. The point of greatest interest in such an ophthalmoplegia consists in the fact that the destructive process has fallen upon nuclei of nerves which are functionally associated, but which are anatomically widely separated, as in the case of the sixth nerve, whose nucleus is located in the pons at some distance below the nuclei of the third and fourth nerves. This destruction of the nuclei of nerves which are physiologically associated is an interesting pathological fact. It shows that because of their functional identity they are equally exposed to some destructive process or to the action of some poison circulating in the blood. In his original paper Hutchinson called attention to the facility with which the progress of the disease is influenced by iodide of potassium, and to the fact that many of these patients had had syphilis, and he was evidently strongly disposed to believe that these cases were of syphilitic origin. In some of his cases the symptoms of locomotor ataxia or some other form of syphilitic paralysis eventually developed.

The *prognosis* of these various forms of ophthalmoplegia depends of course upon their cause. In cases in which they are due to syphilitic infection it is possible, as Hutchinson claims, that an arrest of the process, if not a complete cure, can be obtained. As, however, they sometimes become associated with more widely destructive and degenerative processes the prognosis should always be guarded. They indicate that the nervous system is gravely affected, and the possibility always is that the patient will grow worse instead of better.

The *treatment* of the form of ophthalmoplegia described by Hutchinson is by the iodides and mercury. The effects of remedies in several of his cases, he says, were very remarkable, the patient being rescued from a very dangerous position. He thinks the iodide of potassium is the more valuable remedy, and that it should be pushed for a long period. Relapses may occur, especially if the treatment is not vigorous and long sustained. The progress of the disease, he says, is sometimes arrested, but recovery is never complete. As an example of bilateral partial ophthalmoplegia Hutchinson reported a case of paralysis of both external rectus muscles in a young man following injury to the head. This paralysis did not come on until a month after the injury and was preceded by a severe headache. The left abducens nerve was affected first. The patient held his head constantly turned towards his right shoulder, because he saw best with his right eye. There was no paralysis of any other extrinsic muscle nor of either of the intrinsic muscles of the eye. This patient had had syphilis, and Hutchinson, considering the slow onset of the symptoms, was inclined to believe that the paralysis of the two sixth nerves was due rather to that disease than to trauma. The query arises with reference to such a case whether it might not have been an instance of syphilitic infection arising at the seat of an injury. We know that this may occur, *i.e.*, that the traumatic lesion may become the seat of syphilitic infection in a person who has already acquired that disease. The modern tendency, however, to regard ophthalmoplegia as due to a syphilitic infection of the nuclei would suggest a different explanation, *i.e.*, that the cause of the paralysis was a destructive lesion involving both nuclei of the sixth nerves in the pons rather than the trunks of the nerves. It seems probable, for instance, that the lesion began on the side first affected (the left), and then passed to the nucleus of the other side.

Diseases of the Fifth Nerve.

The fifth nerve, the great sensory organ of the head and face, arises from the largest nucleus or chain of nuclei of any of the cranial nerves and is itself the largest of these nerves. It is the first of these nerves that resembles the spinal nerves, as it has its origin in two roots, namely, a motor and a sensory root; and upon the later or posterior root it has a ganglion.

The nuclei of the sensory branch of the fifth nerve are located for a long distance up and down the pons and the medulla. The motor branch, however, arises from a comparatively small nucleus in the pons. The functions of the fifth nerve are mixed. It is the

great sensory nerve of the head, face, eye, nose, mouth, tongue, and part of the neck. Secondly, by its lingual branch it is possibly one of the nerves of the special sense of taste. Thirdly, by its motor branch it supplies the muscles of mastication, viz., the temporal, masseter, and pterygoid muscles. The superficial origin is from the surface of the pons Varolii about midway between the upper and lower borders. The smaller or motor root is separated slightly from the larger or sensory. The two branches of the nerve pass forward; the larger or sensory root entering a large semilunar ganglion (the Gasserian ganglion), with which the motor root has no connection. As the fibres of the sensory root emerge from the Gasserian ganglion they are divided into three branches: first, the ophthalmic; second, the superior maxillary; third, the inferior maxillary.

The ophthalmic or first main division of the fifth nerve supplies the eyeball, the mucous membrane of the eye and nose, and the skin and muscles of the eyebrow and forehead. The superior maxillary or fifth division of the nerve supplies largely the skin of the upper part of the cheek and the upper teeth. The inferior maxillary or third division of the fifth nerve is distributed especially to the teeth and mucous membrane of the lower jaw, the skin about the ear and temple, the lower lip, the lower part of the face, and through the lingual nerve to the tongue. The motor branch of the fifth nerve is distributed to the temporal, masseter, and pterygoid muscles.

The diseases of the fifth nerve may be divided according as they affect the sensory or the motor root. The former may be subdivided according as they are characterized by abolition of function or by irritation.

Abolition of function or anæsthesia may be caused by any lesion in the course of the fifth nerve or of any of its branches that totally interrupts the conducting function of the nerve fibres. It is not, however, a very common symptom from such causes. Among these causes may be mentioned tumors about the base of the brain or in the orbit; meningitis, especially that which is caused by syphilis; and disease of the bones of the skull. The nerve may also be injured or compressed by disease in the cavernous sinus, by aneurysm, or by tumors or cellulitis in the orbit. Traumata, especially those caused by gunshot wounds, may injure one or other branches of the fifth nerve. Finally, among the most common causes of anæsthesia of the fifth nerve are focal lesions in the pons, such as hemorrhage, tumors, embolic softening, or areas of sclerosis. As, however, the nuclei of the sensory portion of the fifth nerve are spread over such an extensive surface it is not common for all of them to be involved in any one lesion.

Primary neuritis of the fifth nerve is certainly a very rare affection. Gowers believes that it may be caused by exposure to cold. A strictly localized neuritis, such as was recorded by Ziehl, quoted by Gowers, should cause a suspicion of syphilis. Herpes zoster is sometimes observed in the distribution of the fifth nerve and is evidently caused by the same form of inflammation (probably in the Gasserian ganglion) that causes it in other portions of the body.

The anæsthesia due to lesions of the fifth nerve is distributed of course according to the branch involved. Should the whole nerve trunk be involved there will be total anæsthesia in the entire distribution of the nerve. It is more common, however, to have anæsthesia limited to the distribution of one or more branches.

In focal or nuclear lesions of the pons the anæsthesia may be limited to comparatively small and irregular areas. In case one branch is involved, however, the area of distribution of the anæsthesia is strictly demarcated. Thus in orbital lesions the anæsthesia is mapped out strictly in the area of distribution of the ophthalmic nerve on the skin of the forehead, side of the nose, and in the eyeball. The degree of anæsthesia may vary according to the completeness of the lesion; thus in some cases tactile sensibility is only blunted, but in severe cases the sense of touch is entirely abolished. Symptoms of irritation may precede or accompany the early symptoms. Thus there may be neuralgic pains, or burning, tingling, or formication. There may be also other symptoms of hyperæsthesia, as pain on pressure at the points of exit of the nerves. In some cases of extensive anæsthesia of the fifth nerve there is a sort of pseudo-paralysis of the muscles of the face. This is apparently caused by the loss of sensation. It is not a true paralysis of the muscle supplied by the seventh nerve. It is possibly a form of ataxia similar to that seen in other motor regions of the body which are deprived of sensation.

Vasomotor changes may be observed. In the early stages of irritation there may be increased lacrymation and salivation, but in the later stages these two functions are suspended. There may also be flushing of the face or increased pallor. Anæsthesia about the lips and mucous membrane of the mouth causes some characteristic symptoms. When a cup or dish is applied to the lips the patient feels but a portion of it, so that it seems to him as though the utensil was broken. He also chews almost exclusively on the sound side of his mouth, where alone he has tactile sensibility to guide him in the manipulation of the bolus of food. Consequently the anæsthetic side of the tongue may be coated with a thick fur, and this is not a trophic effect, but merely the result of disuse. Taste is affected

on the paralyzed side of the tongue. But much doubt exists as to the course of the taste fibres.

The sense for odors proper is not involved in paralysis of the fifth nerve, as this depends entirely upon the olfactory nerve. Later, however, because of excessive dryness of the mucous membrane the sense of smell may be blunted. Care should be taken to distinguish between odors proper and the more irritating effects of such substances as ammonia and other pungent substances. The sensation for these latter may be entirely abolished in paralysis of the fifth nerve. Magendie, followed by Claude Bernard, believed that smell was a function of the fifth nerve. Romberg properly criticised this view on the ground that Magendie in his experiments had used ammonia and strong cheese and had failed to distinguish between smell and common sensation; the latter was affected by the acrid substances, but this was not a true sense of taste or smell.

Among the most important results of involvement of the fifth nerve are trophic lesions. First of these is herpes. As in other portions of the body herpes zoster follows the distribution of some nerve trunk. In the case of the fifth nerve it may follow any one of the main divisions or main branches. By far the most formidable instance, however, is that which occurs in connection with the ophthalmic nerve. This form is the very grave disease called *herpes zoster ophthalmicus*. It does not differ essentially from herpes elsewhere, but its gravity consists in the fact that it invades and may destroy the delicate structures of the eye. In this disease vesicles form upon the conjunctiva and over the cornea of the eye. These are accompanied with severe neuralgic pains, shooting through the orbit and about the skin of the forehead and temple. In a few days the vesicles turn to pustules. These in turn break down into ulcers or even sloughs. Pus may appear in the anterior chamber of the eye. The iris may become inflamed and adherent and in extreme cases perforation of the cornea may occur. Cases have even been reported, as by Noyes, quoted by Norris,[*] in which cyclitis, followed by shrinking of the eyeball, occurred, and this gave rise by sympathy to inflammation of the other eye. In cases not so grave permanent opacity may result in the cornea, causing more or less impairment of vision. As a rule, tactile anæsthesia of the cornea is present, showing that the sensory neurons of the fifth nerves are seriously involved. According to Norris, the temperature of the skin on the affected side is from one and a half to two degrees above the normal. Anæsthesia also of the skin in the distribution of the ophthalmic branch is usually well marked, though not necessarily absolute. Cases have been reported in which some branches of the ophthalmic nerve have

been involved by herpes zoster without involvement of the eyeball, as for instance the nasal branch. It seems, however, that as a rule this is not the case, as zoster of the nasal branch, *i.e.*, with vesicles along the side of the nose, is usually attended with inflammation of the eyeball (Hutchinson, Bowman). The cause of herpes zoster is probably always some infectious or inflammatory process in the ganglion on the posterior or sensory root of the nerve. (See the discussion of trophic lesions due to nerve injury on pp. 80 *et seq.*) As this ganglion in the case of the fifth nerve is the large Gasserian ganglion we should expect, if opportunity offered, to find evidence of disease in this structure. According to Norris, Wyss and Weidener have found extensive changes. In Weidener's case the Gasserian ganglion, five years after the attack, was found to be shrunken and cicatrized, while in Wyss's case the nerve trunk only two weeks after the onset of the herpes was found thickened, reddened, and surrounded by extravasation of blood from the entrance of the orbit up to the ganglion. These cases prove conclusively that herpes zoster is a disease of the sensory neurons whose cell bodies are located in the large ganglion on the posterior root. They are in accord with cases of zoster in other parts of the body, already referred to, especially that of Charcot and of William G. Spiller. The latter observer has reported to me verbally a case which he saw in Vienna. The patient was suffering with Pott's disease in the cervicodorsal region and herpes zoster was observed along the course of one of the intercostal nerves. At the autopsy the ganglion of the nerve along whose course the herpes had been observed was found macroscopically to be covered with miliary tubercles. This case is a beautiful illustration of the effect of irritating lesions in the ganglia of the posterior roots in the causation of herpes.

Another trophic lesion dependent on diseases of the fifth nerve, but not herpetic in character, is the well-known neuroparalytic ophthalmia. This disease consists of more or less acute inflammation of the structures of the eye and eyeball occurring in cases of paralysis of the fifth nerve. It is usually associated with anæsthesia of the affected structures as well as of other tissues supplied by the fifth nerve or its ophthalmic branch. If this branch alone is involved, for instance, the skin of the left side of the nose as well as the mucous membrane of the left nostril will be insensible. As the inflammation progresses ulcers and sloughs of the cornea may form, perforation results, the iris becomes involved, synechiæ or adhesions bind it to neighboring parts, and ultimately sight may be entirely lost. Norris reports a case of paralysis of the trigeminus, followed by sloughing of the cornea, which resulted in perforation in the course

of a few days. This patient had an epithelioma of the lip and possibly a secondary or metastatic growth within the brain, irritating the fifth nerve somewhere in its course.

While neuroparalytic ophthalmia is always to be feared in paralysis of the fifth nerve, it is not an invariable symptom. Cases are recorded in which after injury or disease the function of the nerve had been abolished for years and yet the eye remained unimpaired.

The course of the affection varies. In one case seen by me at the Philadelphia Hospital an indolent conjunctivitis with excessive lacrymation persisted unchanged for weeks. In some cases, however, as already said, the course is rapid and the destruction great.

The cause of this form of ophthalmia has given rise to much speculation and experiment. Magendie, Longet, and others (see Norris[22]) found that section of the nerve (in rabbits, for instance) causes anæsthesia of the eye and sloughing of the cornea. Section of the nerve anterior to the Gasserian ganglion produces this effect more readily than section posterior to the ganglion. Sir Charles Bell[23] thought that the inflammation was due to the fact that, sensibility being destroyed, the eyelids were not made to close the eye and to wash and clear it, so that inflammation resulted from the irritation of foreign bodies. Graefe, according to Norris, experimented on rabbits and found that section of the fifth nerve caused complete opacity of the cornea, which, however, did not go on to perforation. He consequently held that the change is a genuine trophic change and not due to traumata. On the other hand, Snellen found that when he cut the fifth nerve in rabbits and protected the eye the cornea remained intact for at least ten days. The proper explanation of this change in the eyeball is probably that it is a true trophic lesion dependent on disease or injury to the peripheral sensory neurons. It is thus strictly analogous to the trophic lesions, such as pemphigus, whitlow, and herpes, which are seen in other portions of the body, as in the hand, arms, legs, and feet, following upon nerve injury. Magendie's observations that section of the nerve in front of the Gasserian ganglion is more likely to produce this effect than section posterior to the ganglion are clearly confirmatory of the theory that the lesion is trophic. Section in front of the ganglion cuts off the sensory arborizations in the mucous membrane and the eyeball from their trophic centres, which are the cell bodies of the neurons in the Gasserian ganglion. The fact that all cases of fifth-nerve paralysis are not followed by a neuroparalytic affection is probably confirmatory of this view. Lesions which irritate the Gasserian ganglion itself, *i.e.*, the cell bodies of the sensory neurons or the dendritic

branches of these neurons (the fibres passing from the ganglion to the periphery) are probably much more likely to cause these acute irritative trophic changes than are lesions in other portions of the trunk of the fifth nerve, *i.e.*, posterior to the ganglion.

Althaus[44] has recorded an interesting case of paralysis of both fifth nerves in a young man, apparently the result of exposure to cold. The first symptom was pain. This yielded to treatment apparently, but after it had gone the cornea of each eye became covered with thick opacities, which resulted in complete blindness in the right eye and partial blindness in the left. The power of mastication was lost and the skin of the face and the mucous membranes became quite numb. There was light œdema of the cellular tissues of the face which gave the man a peculiar statuesque expression. There was no paralysis of the facial muscles. The sense of smell was not affected. All the intrinsic and extrinsic muscles of the eyes were unparalyzed. The several modes of sensation were abolished—temperature sense, tactile sense, and the sense of locality. The mucous membranes of the eyes, nose, and mouth were also anæsthetic. Reflex movements in the eyes, as well as lacrymation upon irritation of the mucous membrane, were abolished. There was, however, hypersecretion of mucus in the conjunctiva and in the nose. That from the nose was so acrid that it irritated the skin of the lip. The growth of hair on the head and face was not affected. Snuff did not cause sneezing. The tongue was anæsthetic and had been frightfully bitten and lacerated by the patient without his knowledge. The secretion of mucus in the mouth was so excessive as to oblige the patient to use a handkerchief constantly in order to catch it. Extracting a tooth did not cause pain. The teeth, however, were not the seat of trophic changes. The sense of taste was not impaired on the back part of the tongue; in the anterior part of the tongue, however, it was blunted. The muscles of mastication were paralyzed. The patient had a continuous rushing noise in the head, the cause of which was obscure. There was no history of syphilis.

The following case of paralysis of the fifth nerve was under my observation in the nervous wards of the Philadelphia Hospital recently. It has been reported by Charles K. Mills.[45]

L. D——, aged 26 years, a mulatto, was probably syphilitic. Four months before admission he began to have headache, both frontal and occipital. One morning the right side of his face was found entirely paralyzed, with ringing sensations in his ear. On admission he had complete paralysis of all the facial muscles supplied by the seventh nerve and of the muscles supplied by the motor division of the fifth nerve. Anæsthesia was present on the right side of his face, extend-

ing from the median line to the angle of the jaw and from two inches above the hair line on the scalp to the point of the chin. The conjunctiva of the right eye was totally anæsthetic as was also the right half of the tongue. Taste was abolished on the right side of the tongue both in its anterior and posterior portions. Reaction of degeneration was observed in the affected muscles. A slight neuroparalytic ophthalmia was present in the right eye, as shown by a slight haziness which had spread over the cornea. In this case large doses of potassium iodide caused improvement. The anæsthetic area decreased, the ophthalmia passed away, and the paralyzed muscles regained some power. The cause of the condition was probably a gumma or a syphilitic meningitis on the lateral aspect of the pons.

FIG. 21.—Paralysis of the Fifth Nerve. (Philadelphia Hospital.)

Another case from the same clinic, also reported by Mills,[88] was an instance of combined paralysis of the fifth and seventh nerves. (See Fig. 21.) This patient probably had brain syphilis. He had had recurring attacks of paralysis of the left half of the body. The last attack left him with complete facial palsy on the left side. In addition he had paralysis of the motor division of the fifth nerve and limited areas of anæsthesia in the distribution of this nerve. The ophthalmic branch especially was involved and the patient had a high grade of neuroparalytic ophthalmia, for which it had been found necessary in the hospital to enucleate the eyeball.

The second class of sensory affections of the fifth nerve comprises those that are characterized by irritation. These may be divided into headache, neuralgia, migraine, and tic douloureux. All these diseases are properly described here except migraine.

Headache.

Headache is rather a symptom than a distinct disease. Its exact pathology is obscure in any case, but as it is an acute pain occupying some position in the area of distribution of the fifth nerve it is reasonable to suppose that its seat, at least in some instances, is in that nerve. It may be due to irritating substances in the blood or to vasomotor changes affecting the circulation in the nerve branches or in the meninges of the brain or even in the brain itself. However caused, headache probably finds expression as a pain by irritating the branches of the fifth nerve distributed within the cranium. It must not be forgotten, however, that the meninges receive numerous filaments from other nerves, especially the sympathetic, and that these may also be the channels of the pain sense.

Headache is such a common symptom of various disorders of health and is so little likely to follow definite laws that it is difficult to describe it systematically. It occurs at all ages and in both sexes. According to Dana[87] the most common ages for headache are from ten to twenty-five and from thirty-five to forty-five years. It is very uncommon in early childhood and in old age. Women suffer more than men and it is probably rather more frequent among the wealthier classes.

The causes of headache are various. Among the most common are the various toxæmias. Gout and rheumatism are not infrequent causes of headache. In gout especially this symptom is important and too often overlooked. The headache of gout is usually paroxysmal and it may even substitute an explosion of the disease in some other portion of the body. Thus in a gentleman recently under my observation severe and obstinate frontal headache was the only symptom for a long period. He had been subject formerly to attacks of gout in the great toe and during this attack of headache had uric acid in excess in his urine. He one day wore a tight-fitting shoe with a fold of the stocking making pressure across his formerly gouty joint. This caused an explosion of gout in the joint with immediate and permanent relief from the headache.

Uræmia not uncommonly causes headache. In fact in cases in which persistent headache is a new and obscure symptom this cause should always be searched for. The severe headache of uræmia is sometimes associated with nausea and vomiting, the complex of symptoms thus simulating a gastric disorder. A paroxysm of headache may precede and herald in a convulsive attack or a state of coma. In the blood poisoning of puerperal eclampsia headache is

not an uncommon symptom and is sometimes a very early and suggestive one. In this state it may be associated with amblyopia or obscurity of vision or visual hallucinations, as of moving objects, specks or clouds, or bright light before the eyes.

In diabetes headache with gastric disturbance, followed by delirium and dyspnœa, is often one of the preliminary symptoms of one form of diabetic coma.

In the various infectious fevers headache is not uncommon. In the early stages of typhoid fever this symptom is often urgent and distressing.

The various toxic substances, such as alcohol, lead, and tobacco, not infrequently cause headache. In lead poisoning this is not, however, a very frequent symptom. In that grave complication, fortunately somewhat rare, called "lead encephalopathy," an excruciating headache is often the prominent symptom. This is associated with or followed by delirium or stupor, passing into coma.

In acute alcoholic poisoning severe headache is a frequent sequel, while in chronic cases the toper's morning headache is well known.

The various severe neuroses, such as hysteria, neurasthenia, epilepsy, and migraine, frequently cause headache.

The headache of hysteria is quite characteristic. A common form is the *clavus*. This is a circumscribed pain in the head of very limited extent which has been likened to a pain such as would be produced by driving a nail into the part, hence the name. This clavus may be accompanied with ringing and beating noises in the ear and with the feeling of being beaten on the temple with mallets. This symptom, as already said, is conspicuous among the prodromes of the grand attack of convulsive hysteria.

In neurasthenia headache and also pains in the back and limbs are not uncommon. These pains are rather diffused and are not as a rule very intense in character. They are often associated with a sense of fatigue, even on slight exertion. The headache of neurasthenia is not, as a rule, diffused over the entire head. Its favorite seats are the occiput and the brow just above the eyes. When located in the occiput it is likely to be associated with pain in the back of the neck and between the shoulders. The headache of neurasthenia is sometimes characterized by the sense of a tight band drawn about the head. Headache is very readily caused in some neurasthenic patients by any effort, whether mental or physical, however slight. In some rare cases it is a severe and obstinate symptom.

The headache of epilepsy is an important symptom. It almost invariably follows the attack and may persist for some hours or even days. Sometimes the headache, with the associated sensory symp-

toms, constitutes the whole of the attack and may consist in a crisis resembling migraine. Some authors, in fact, have tried to point out an analogy between migraine and epilepsy, calling attention especially to the explosive character of these two neuroses, but a relationship is not satisfactorily proven. Certainly ordinary migraine does not show a tendency to pass into motor epilepsy. In these cases of epilepsy, in which the headache and sensory symptoms are prominent or isolated, the attack may be considered substitutional, *i.e.*, the sensory crisis substitutes the motor or convulsive crisis. This may be one form of what has been termed "masked" epilepsy.

In migraine headache is one of the essential symptoms of the paroxysm. It is usually ushered in by some affection of the optic nerve, such as amblyopia, or various forms of visual hallucination. As these pass off, usually after a few minutes or an hour, the headache gradually supervenes. It is often very severe in type and may endure for as long as a day or even two, completely prostrating the patient. The attack usually closes with nausea and vomiting. Migraine is essentially a paroxysmal disease and may possibly be caused by some autogenous poison in the blood. The attacks vary in intensity and frequency according to individuals. It is most severe in childhood and early adult life and tends to abate in middle life and frequently ceases entirely before old age.

Another class of causes of headache may be considered reflex. Among the most common of these is eye strain. Errors of refraction not infrequently cause headache. The pain may be either frontal or occipital; very rarely it is more diffused over the head. The patient not infrequently is ignorant of the true cause of this symptom. Its character is often indicated by the fact that it is brought on or intensified by over-use of the eyes at fine work, such as reading, sewing, and embroidering. Another ocular cause for headache, although not truly reflex, is glaucoma. It may be an early symptom, rather paroxysmal in type, of extreme intensity, associated with paleness of the face, nausea, and vomiting. In chronic cases it may also be paroxysmal, very intense, simulating to some extent migraine. In these cases close inspection will reveal increase in the intraocular tension, irregularity in the pupil, with a sluggish iris, anæsthesia of the cornea, haziness, and impairment of vision.

Diseases of the naso-pharynx may cause pain in the distribution of some branch of the fifth nerve. The pain may be especially in the temporal region or in the forehead. It may be accompanied in extreme cases with nausea. It is not, however, truly reflex in character, but is usually due to direct irritation of the trunk of a nerve. Inspection of the nose will usually reveal the true cause of the symptom.

Dyspeptic disorders are associated at times with headache. There is nothing especially characteristic about this symptom. It is possibly due to impoverished or poisoned blood, the result of gastric disorder.

It is commonly said that disorders of the sexual system may produce headache. Masturbation, especially when associated with a characteristic neurasthenia that develops as a result of this habit, may cause headache and aching pains in the back. This headache is especially likely to supervene immediately after the act, and may last for some hours. In confirmed cases it may persist longer, and is an expression then of a general depraved state of nutrition of the blood and nervous system.

Finally, common causes of headache are found in various organic diseases of the brain and of the cranial bones. Tumor of the brain is very commonly accompanied with headache. In fact, this symptom is rarely absent, and in most cases it has peculiar characteristics (Mills and Lloyd[66]). It is usually a continuous pain of great severity, but with exacerbations or paroxysms of great violence. In some cases, however, it is dull or throbbing, accompanied with a sense of weight, pressure, or constriction, but even in these cases it usually increases towards the end and becomes less endurable. In the table of one hundred cases of brain tumor collected by Mills and Lloyd, headache of some type was present in sixty-six. In only five cases was it stated not to have been observed, while of the twenty-nine cases in which no mention was made of it, it was probably present in a large majority. In some cases the pain seems to be localized at a point near the tumor. It may even be associated, especially in the cases of tumors involving the meninges, with tenderness over the skull. In cerebellar tumors the pain is sometimes occipital, but this is not an invariable rule. Other organic lesions of the brain that cause headache are meningitis, especially syphilitic, aneurysm, and diseases of the cranial bones involving the meninges and the brain cortex.

Neuralgia.

Neuralgia may be defined as a pain, paroxysmal and intermittent in character, confined to one or more branches of a nerve. It is not uncommon in the fifth nerve; in fact, this nerve is one of its favorite seats. Its main characteristic, as indicated by its name, is pain. It has few if any objective symptoms. The distinction between headache and neuralgia is rather vague and arbitrary. Neuralgia is usually a more circumscribed affection than headache, *i.e.*, it is more likely to be limited to one or to a few nerve trunks. The underlying

condition of the nerve structure in neuralgia is obscure in many cases. The disease, as already said, is usually paroxysmal. It is possibly due to some irritant circulating in the blood, or it may in some cases be simply an inflammation of the nerve trunk or a neuritis. It is customary to describe neuralgia as being either idiopathic or symptomatic, by which is meant in the first case that the disease is purely functional, and in the second that it is due to an organic change in the nerve itself. Such a distinction is of little value; in fact, it is entirely misleading. The severe pain of a neuralgia cannot be caused by anything less than some irritating agent affecting injuriously the sensory neurons. That this agent is often obscure and even paroxysmal in its action does not furnish a ground for the claim that the disease is purely functional. The most typical neuralgias are those that occur paroxysmally in some branch of the fifth nerve. These paroxysms may be comparatively short in duration, or they may last for days or even weeks. Not infrequently the intermissions are long, although the rule varies in this respect. The most common causes of neuralgia are doubtless toxic states of the blood; thus rheumatism, gout, malaria, syphilis, diabetes, and other dyscrasias are especially likely to cause neuralgia. Heredity seems to play a part in some forms of neuralgia. Victims of the disease are not infrequently neurotic or of neurotic stock. Reflex causes (so frequently assigned to neuralgia) are probably exaggerated. Eye strain, as already said, not infrequently causes localized headaches, which might, of course, be with propriety called neuralgic, for after all the use of the word "neuralgia" is somewhat arbitrary.

Neuralgia is usually a disease of adult life. In the case of children neuralgic attacks should always excite the suspicion of migraine. In persons well advanced in adult life the onset of neuralgic attacks should excite the suspicion of some organic cause for them or of some dyscrasia. Thus syphilis, gout, rheumatism, malaria, uræmia, diabetes, glaucoma, eye strain, disease of the teeth or of the nasal chamber, or some commencing organic disease within the cranium should all be searched for.

The pain of neuralgia is usually of a severe type. Cases occur of course that are an exception to this rule, but the majority of patients describe the pain as peculiarly harassing and unendurable. It is usually sharp and lancinating in character. This is so especially in the fifth nerve, the neuralgia of which usually presents a type of great severity. Even during the paroxysm the pain, however, is not always constant. It may remit only to recur again with renewed intensity. There is sometimes even a distinct periodicity in the occurrence and recurrence of the exacerbations. This was formerly sup-

posed to indicate a malarial origin for neuralgia, but this view no longer prevails. Exposure to cold during a paroxysm usually increases its severity. Firm pressure often relieves, but a light touch may aggravate the pain of a neuralgia of the fifth nerve. In very severe cases the skin may become flushed and the eyes slightly suffused; or, on the other hand, the face may show an unusual pallor.

The pathology of neuralgia consists probably in some slight, more or less temporary changes in the neurons, due to the circulation in the blood of some toxic agent. These changes may amount in some cases to a slight inflammation or neuritis. This is so especially in cases of the most persistent character and most severe grade. In case a distinct neuritis were the underlying condition of an ordinary neuralgia it is likely, however, that we should have other and objective symptoms. Thus anæsthesia, paralysis of motor fibres in mixed nerves, and trophic changes should occur. The fact that these are seldom if ever present in ordinary paroxysmal facial neuralgia is rather against the theory of such a distinct organic change as inflammation being the cause of the symptoms. The most acceptable theory in my judgment is that in most cases of neuralgia the pain is the expression of an irritation of the sensory neurons caused by toxic substances in the blood, and that this irritation in the great majority of cases does not proceed to a true inflammation. In cases in which a distinct neuritis can be demonstrated, as, for instance, by the swelling of the nerve trunk at the supraorbital notch, as claimed by Sinkler, there does not appear to be any reason why the case should not be classed as one of neuritis instead of being especially designated as neuralgia.

From what is said it appears that the main points for the diagnosis of neuralgia are severe pain, paroxysmal in character, and limited in its area, and the absence of symptoms of profound organic change in the nerves, such as anæsthesia, paralysis, and trophic changes.

Migraine.

Migraine is one of the explosive neuroses. Brief mention for the sake of comparison may be made of it here. Migraine is more than a mere affection of the fifth nerve, although one of its most severe symptoms is located in that nerve. It is characterized by visual, neuralgic, and gastric symptoms, and although usually described as a mere neurosis, it is possibly due, as already explained, to some autogenous toxin in the blood. The pain of migraine is located as a rule—to which there are few, if any, exceptions—in the head. The attack is usually ushered in by a sensory aura. In the vast ma-

jority of cases this aura is in the nerve of sight. It is usually an amblyopia, although in some cases scintillations, spectra, phantasmagoria, undulatory, wave-like, or rotating lines or currents, and in rare cases the figures of animals or men are seen. In some cases, however, the sensory aura may be felt in other nerves of the body, as for instance a tingling or numbness in the hand, arm, or shoulder. These auræ subside before the onset of the neuralgic pain. This pain is usually severe in character and may persist for hours or even for days. The attack terminates with gastric disturbance, such as nausea and vomiting. Rachford, quoted by Wilson," attributes migraine to a leucomain poisoning and has found paraxanthin and xanthin in the urine.

Migraine frequently begins in childhood, reaches its height in early adult life, and declines after the meridian of life. It is to be distinguished from other forms of headache and neuralgia by its peculiar constitutional and paroxysmal type, by its auræ or prodromes, and by its gastric disturbances.

Tic Douloureux.

This dreadful disease is a special form of neuralgia of the fifth nerve. It is characterized by pain, usually of the most intense character, located at first in one or perhaps more branches of the trigeminus. It is not so distinctly paroxysmal as the other neuralgiform affections of this nerve. It is, however, subject to severe exacerbations and to periods of very marked remittence. In some cases, however, the pain is practically constant, and in no case, as a rule, does it completely subside. The tendency for tic douloureux to be located in only one trunk of the fifth nerve is a marked characteristic. It is, moreover, a disease of adult life, the great majority of cases being in persons over forty years of age. It is seen about equally in both sexes.

The *causes* of tic douloureux are altogether obscure. Regarding the disease, as I do, as a degenerative one of the sensory neurons, especially their cell bodies in the Gasserian ganglion, I feel that it has not yet been practically demonstrated what the exciting cause of this irritative degeneration is. The histories of cases seem to indicate that the disease is sometimes due to trauma, exposure to cold, carious teeth, or various debilitating agents. These may and probably do in some cases act as exciting causes, but, considering the inveteracy and the severity of the symptoms, it seems more than probable that the proximal or underlying cause must be some deeper seated degenerative tendency in the bodies of the neurons themselves. Syphilis and

the various dyscrasiæ caused by gout, rheumatism, and malaria cannot with certainty be held responsible for the larger proportion of cases of tic douloureux. Heredity is an unimportant factor, and the over-use of alcohol and tobacco does not appear to be an element in the causation of this disease.

The *symptoms* of tic douloureux are intense pain, lacrymation, flushing of the face, and rarely spasmodic movements of the muscles supplied by the seventh nerve. Of these symptoms the pain is the only constant and characteristic one.

The pain of tic douloureux is peculiarly severe and atrocious. It is a stabbing or darting pain, occurring as a rule in short paroxysms of great intensity but not completely remitting. These paroxysms are excited by the most trifling causes, such as exposure of the face to cold air, movements of the facial muscles or of the tongue, attempts to drink or to chew food or to talk; in many cases, in fact, paroxysms occur without apparent exciting cause. Sometimes they are excited by movements of other or distant parts of the body, such as slight muscular exertion, attempts at dressing, arising from the recumbent to the sitting position, or combing or brushing the hair. In cases in which the tendency to these paroxysms of stabbing pain is the greatest the patient in fact is often forced to lie supine, dreading to move, to speak, or to take food.

The paroxysms of tic douloureux may or may not be associated with vasomotor phenomena. As a rule, in cases which I have observed, no such phenomena have been seen. Lacrymation is occasionally seen, although it is by no means common or characteristic.

Spasmodic movements of some of the facial muscles are sometimes symptomatic of tic douloureux. These movements are seen more especially in cases in which the maxillary divisions of the fifth nerve are involved, and in that type of case especially which has been miscalled *epileptiform*. In this type the stabbing pains occur with great suddenness and, as it were, with lightning-like rapidity, and the facial muscles are thrown into spasms as a result. These spasms, in some cases, are possibly not entirely reflex. They may be in part volitional, the patient fixing the muscles of the face in an attempt apparently to immobilize the parts so as to remove all sources of irritation from the branches of the nerve. In some cases, however, these spasms in the facial muscles appear to be truly reflex. The muscles in groups are thrown into tonic or clonic spasms over which the patient has no control, these clonic spasms themselves seeming to excite in turn renewals of the explosions of pain.

The location of the pain of tic douloureux varies. As an almost invariable rule, however, this pain in the early stages of the disease

has rather a strictly limited localization. Thus it is not uncommon to see only one nerve trunk involved, and this may be the only branch of the fifth nerve affected for a comparatively long period. This is so common that it may be considered almost a characteristic of the disease. As the case progresses, however, other branches of the same division become affected, and ultimately the pain may pass from one main division of the fifth nerve to the other.

In a case in which the ophthalmic division is involved the pain is felt chiefly in the supraorbital branch. It is felt especially at the supraorbital notch, where the nerve emerges from the skull. From this point the pain follows the ramifications of the nerve upon the brow. The pain is also felt along the side of the nose following the nasal branch, and in some cases it is deep within the orbit. Pain on pressure is felt especially at the points where the branches of the nerve emerge, hence at the supraorbital notch, at the upper side of the nose near the inner canthus of the eye, and at the point of emergence of the nasal nerve on the side of the nose. This type of tic douloureux has been supposed (without sufficient reason) to be an evidence of malarial poisoning.

In cases in which the superior maxillary division of the fifth nerve or its branches is involved the pain is felt with especial severity on the upper part of the cheek. This is a not uncommon type of tic douloureux. The branches of the superior maxillary nerve emerge upon the face at the infraorbital foramen. From this point the pains of tic douloureux radiate, involving the cheek, the median portion of the side of the nose, and the whole of the upper lip. The tender points are especially at the infraorbital notch, where the nerve emerges beneath the orbit, at the side of the nose, and over the malar bone.

In cases in which the inferior maxillary division of the fifth nerve is involved the pain is rather more extensively diffused, because of the greater number of branches of this division and their wider or more extensive ramifications. The pain is felt about the skin of the chin and lower lip, lower jaw, cheek, anterior portion of the ear, temporal region, teeth and gums of the lower jaw, and side of the tongue. The most common type of tic douloureux in this location is that in which the inferior dental branch is involved. This branch of the inferior maxillary division of the fifth nerve is the largest. It lies embedded in the dental canal of the lower jaw bone, sending branches to the lower teeth and emerging from the canal at the mental foramen upon the chin. Its branches supply the skin and mucous membrane of the lower lip and the skin of the chin as well as some of the neighboring muscles, which thus receive their common and muscular sensi-

bility. At this point of exit at the mental foramen the inferior dental nerve is especially sensitive in some types of tic douloureux. In one instance I knew it to persist thus localized for a period of several years. A space hardly as large as a silver dollar surrounding the mental foramen and including the lower lip was the seat of most exquisite pain, inhibiting all movements at times of the lip and lower jaw. In time, however, the auriculotemporal nerve became involved so that shooting pains were felt about the pinna of the ear and the temporal regions; later still the lingual or gustatory branch was involved so that the paroxysms of darting pain were felt along the side of the tongue. This characteristic commencement and distribution of pain in the branches of the inferior maxillary nerve were evidences probably of the central or ganglionic origin of the affection.

As an almost universal rule structural changes are not seen in cases of tic douloureux. Anæsthesia in the distribution of the affected nerve is rare but not unnoted. True trophic changes, such as herpes and pemphigus, have not, so far as I know, been observed. Vasomotor changes, such as flushing of the face and lacrymation, are occasionally seen.

Inhibition of the movements of the face, lips, and tongue, and of the muscles of mastication is very common in this disease. This becomes in fact in severe cases a grave complication. I have known patients to refuse food for many hours because of the agony experienced in attempting even to draw fluids into the mouth, and to take so little during a period of many days that the strength was seriously undermined and the prospects for recovery correspondingly diminished.

The various types of tic douloureux have been sufficiently indicated in the foregoing descriptions. The most severe form of the disease is that in which with more or less continuous pain there occur paroxysms of great suddenness and intensity. This type has been called epileptiform. That type in which reflex or associated spasms of the facial muscles occur has been called convulsive tic. The various types according to location are the supraorbital, infraorbital, nasal, malar, mental, and lingual. These types are simply due to variations in the location and intensity of one and the same pathological process.

The *pathology* of tic douloureux is still a subject of some obscurity. It has been customary to state that the underlying cause is inflammation of the affected branch of the nerve. Some anatomical proof in support of this theory has been adduced, as, for instance, a case reported from the Orthopædic Hospital, Philadelphia, in which a nerve trunk excised by Dr. Keen presented under the micro-

scope the signs of interstitial neuritis. Considering the peculiar onset and progress of tic douloureux I have long been led to believe that the irritative process, whatever it may be, whether inflammatory or more truly degenerative, has its seat probably in the Gasserian ganglion. This ganglion is composed of the cell bodies of the sensory neurons which compose the fifth nerve. It is exactly analogous to the ganglia on the posterior roots of the spinal nerves. An irritative process having its origin in a small group of these sensory neurons would cause pain, which of course would be referred to the distribution of these neurons on the skin or to the course of their irritated axis cylinders in the nerve trunk. Such a process would probably involve at the beginning only a very small group of contiguous cells in the Gasserian ganglion. These contiguous cells would probably constitute a group, sending their axis cylinders to one particular nerve trunk. This would account for the strictly local character of the pain at the commencement. As the irritative process advanced other cell groups would be involved, and thus gradually other nerve trunks would begin to transmit painful impressions. As the cell bodies of the neurons, which are the nutritive centres, degenerated, the axis cylinders would also undergo destructive changes, according to the well-known law of degeneration. In the course of time proliferation of interstitial tissues in the nerve trunks would occur and thus many of the appearances of an inflammation might be presented. This practically is what has been found in the excised branch of the fifth nerve. Therefore the mere fact of the presence of degenerative or inflammatory changes in the excised nerve does not prove that the initial process was in that nerve and not in the ganglion. The fact that cure or relief has been gained temporarily by excision of the nerve trunk can be explained on the supposition that the powerful impression made upon the neuron by totally ablating its axis cylinder might for a time arrest the inflammatory or degenerative process in its cell body or impair its transmitting power. But even admitting that this supposition is unwarrantable, the fact remains that in almost all cases in which the affected nerve trunk has been excised the disease process almost invariably passes in time into another nerve trunk, and this certainly could only be by way of the Gasserian ganglion.

Again, this process of a slowly degenerative and irritative change passing from cell body to cell body within the ganglion itself is strictly analogous to what we see occurring on the motor side of the nervous system, *i.e.*, in progressive chronic anterior poliomyelitis. On the sensory as on the motor side the change seems to pass from cell body to cell body, the characteristic difference being that in the

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latter case we have progressive paralysis and muscular atrophy and in the former progressive neuralgic pain.

The fact that tic douloureux in the majority of instances is seen in persons well advanced in life might be looked upon as presumptive evidence that the disease process is one of degeneration. But this evidence is not of great value from the very fact that many cases occur in comparatively young people.

Trophic changes, as already said, are comparatively unknown in tic douloureux. J. F. Walsh [°] reports a case of neuralgia confined to the lingual branch of the fifth nerve in which after stretching that branch the tongue appeared slightly pushed over towards the affected side, but there was no atrophy or other trophic change. In this case, which occurred in a young woman twenty-six years of age, the pain was entirely confined to the right lingual nerve. It is rather difficult to understand, considering the severity of the irritative process in tic douloureux, why trophic changes are not more common. The fact that they are not seems to militate against the theory that the changes in the Gasserian ganglion are of an acute inflammatory character. We know that inflammatory changes of an acute kind in the intervertebral ganglion, and in the nerve leading from it, not infrequently cause herpes zoster, and we have seen in this article how the irritation in the ganglion caused by small tubercles will be followed by an herpetic eruption. Nothing like this apparently ever occurs in ordinary tic douloureux. The inference seems warrantable, both from this fact and from the extreme slowness and chronicity of the disease, that the change in the ganglion is not one of an acute irritative character, but rather of some slow degenerative process. Additional light upon this important subject must be supplied by microscopic studies on the ganglia that have been excised.

The following case of tic douloureux is at present under my care in the Philadelphia Hospital:

P. T——, 58, white, occupation weaver, has had a high grade of convulsive tic for six years. Formerly the spasms varied in frequency from every five minutes to several hours. They are brought on by eating, drinking, and by sudden changes in the temperature. The pain at first was worse on the cheek and temple. During a spasm the face becomes flushed, the muscles twitch markedly, and the pain is excruciating. The patient grasps his face in his hands and begins to tremble all over and has an expression of great agony on his face. On admission to the hospital this patient admitted an addiction to alcohol but denied that he had ever had any venereal disease. His present condition is as follows: His hair is gray, more so on the affected side of the scalp; the nose is much deformed as the result of a severe accident, which caused fracture of the

bone and deviation of the nose to the right; the nasal chamber of the left or affected side is almost obliterated by the deviated septum and the deformity of the bones; the nasal mucous membrane has a purplish hue, and shows evidence of a chronic inflammation. The man's arteries are firm, sclerosed, tortuous, and of a very high tension. The paroxysms of pain and associated spasms occur now every few moments and are excited especially when the patient attempts to talk or to take food. These spasms involve the left occipitofrontalis muscle and the muscles of the cheek and upper lip. The pain is of a darting character and excruciating in degree. It is most marked during the spasm just above the external canthus and in the region of the supraorbital nerve. There is also considerable pain at the infraorbital foramen and on the left side of the upper lip, also at a point about an inch below the left zygoma whence it runs forward to the alveolar border of the superior maxillary bone of the same size. In this case the spasm of the muscles is tonic and not purely reflex, because it can be controlled to some extent by the patient. It seems rather to be an effort on the part of the patient to make the face and all the painful parts immobile with the instinctive hope or intention of thus reducing the stabbing pain to a minimum. The relationship of trauma to the onset of tic douloureux in this case is most interesting. The man had a severe accident which caused fracture and great deformity of the bones of the nose, and this was followed in a short time by commencing tic douloureux. It is a question whether the original wounding of the nerve endings may have been the starting-point for an irritative process, inflammatory or otherwise, which then ascended to the Gasserian ganglion and later spread to the other neurons. The distribution of pain now is widespread through the branches of the ophthalmic and superior maxillary divisions of the fifth nerve. This patient has already submitted to an excision of the nerve at the infraorbital foramen, but the operation gave no relief, probably because the pain was not limited to this nerve.

The medical *treatment* of tic douloureux is most unsatisfactory. All the sedative and alterative drugs known to medical science have been used upon this obstinate disease and not one of them has made a permanent reputation. The risk of formation of some drug habit by these unfortunate patients is very great. This is true especially in reference to morphine and other opiates and cocaine. It is possible with these and similar anodynes to give the patient temporary relief, but their repetition is so soon, so frequently, and so urgently demanded that the physician must early come to feel that he is standing before the ugly alternative of refusing to relieve his patient on the one hand, or of enslaving him to a drug on the other. It is scarcely worth while to mention the whole list of sedative and anodyne drugs. Morphine or some opiate will undoubtedly give the promptest and most efficient relief for a short time from the severe paroxysms. Cocaine by injections is also an efficient as well as seductive drug. The chances of

the patient coming to rely upon it are rather greater even than in the case of morphine. I have thought in some cases that morphine in small frequently repeated doses, combined with alteratives and tonics, such as iron and quinine, was a curative and not merely palliative drug. It is better used thus than in larger doses at less frequent intervals. Its steadily anodyne influence upon the irritated nerve when given in small repeated doses, as for instance gr. $\frac{1}{12}$ three or four times a day, seems to have a beneficial effect. Its combination with strychnine is especially valuable. This latter drug may be used also in regularly repeated doses, as recently recommended by Dana. Caution must be observed, however, not to use too large a dose, for the drug may act as an irritant to the nerve. Dana seems to think that it acts in some special way in tic douloureux. This may be true, for it may possibly promote nutritive and alterative changes that are beneficial. Its combination with small doses of morphine is advantageous because the latter tends to control any irritative effect of the former. Nitroglycerin in doses of gr. $\frac{1}{100}$ frequently repeated is sometimes useful. The only other drug that seems to enjoy anything like a permanent reputation in the treatment of tic douloureux is aconitine. It may be given in doses of gr. $\frac{1}{130}$ to $\frac{1}{100}$. Bromide of potassium is not a useful drug in tic douloureux or any other painful affection, because it is not an anodyne. Its disagreeable constitutional effects are incurred without any compensation. The antipyretics, such an antipyrin, antifebrin, exalgin, phenacetin, and a proprietary drug known as antikamnia have a controlling influence in some of these cases. In very severe cases, however, I have found their use unsatisfactory both because of their slight and evanescent effects and also because their continuous use exercises an injurious effect upon nutrition. They tend to produce anæmia and so otherwise disturb nutrition, and a tolerance to them is soon established by the patient which renders their analgesic effects less and less marked. Chloroform and ether in sufficient doses will of course deaden pain, but their effect is strictly temporary. Of other sedative drugs mention may be made of belladonna, Indian hemp, and croton chloral. Belladonna and its alkaloid atropine are not satisfactory for the reason that their analgesic effects are not great and their constitutional effects, especially the dryness of the throat, are peculiarly objectionable in a disease in which the difficulty of maintaining the secretions of the mouth and throat in a satisfactory state and of administering food is already very great. Indian hemp or hasheesh has been highly lauded, but its intoxicating effect upon the brain (especially in large and repeated doses) is much against it. Croton chloral is undoubtedly of service, especially when

combined with the small repeated doses of morphine already mentioned. Alterative drugs such as arsenic and iodide of potassium have never seemed to me to be of much service in any form of neuralgia of the fifth nerve. The same may be said of the mercurial preparations. Syphilitic affections of this nerve are certainly rare, but in cases in which syphilis is demonstrable mercury and the iodides would probably be efficacious. Great care should be observed in all cases of tic douloureux to maintain the nutrition and strength of the patient. Consequently the selection of an appropriate diet and the administration of a proper quantity of food should be among the first cares of the physician. Small doses of some alcoholic stimulant, especially a good wine or whiskey, are generally indicated.

Local applications are frequently comforting in this disease. The sedative action of ice should be tried. The ice should be used in a small rubber bag properly protected by several thicknesses of flannel. On the other hand, warm or almost hot applications are sometimes useful. The various sedative and anodyne liniments, such as chloral and camphor, or those containing chloroform and aconite, are not without use, although in many instances they are disappointing. Electricity, especially the constant current, may be tried; it is sometimes not without good effect. But great care should be observed to use a mild current with the anode over the painful point and to avoid shocks. Faradism may be irritating in any form of neuralgia of the fifth nerve. The cataphoric action of electricity is sometimes useful. By this action a sedative drug may be diffused through the skin over the painful area. The best drugs for this purpose are cocaine, chloroform, and tincture of aconite. They are applied best by saturating a piece of tissue paper or a thin layer of lint with the remedy, and the positive pole with a weak current is then applied upon the paper or lint. By this means some diffusion of the medicinal substance is caused and the relief may be prompt and may endure for some hours. The removal of carious teeth may be proper in cases in which the dental nerve seems to be the seat of irritation. The indiscriminate extraction of a large number of sound teeth, however, is to be condemned, for it is too great a price to pay for what is usually a most disappointing result.

General hygiene is of great importance in all of these cases. In one particularly obstinate case I knew great and permanent benefit result from a sojourn during the winter months in one of our Southern States. Travel, however, is very irksome, irritating, and debilitating to some of these patients, and the physician has to take into due account the possible disadvantages of a long journey, removal from the comforts of home, and separation from regular

medical and other attendance which are involved in such a radical change. Fresh air, especially outdoor exercise, while theoretically advantageous, is practically often impossible for a patient suffering with the torments of tic douloureux. If a removal from home is made it is best to select a dry and warm climate. The cold and dampness of the seashore are not as a rule beneficial to these patients.

The radical and only successful treatment in some cases of tic douloureux is surgical. In cases in which the pain is limited to only one branch, that branch alone of course need be excised. The particular branches which in my observation are most likely to require excision are the supraorbital, superior maxillary, inferior dental with its mental branch, and the lingual. Appropriate operations can be performed in all these cases and are sometimes followed by most gratifying temporary results. The permanent results, however, in too many of these cases are disappointing. The tendency of the disease undoubtedly is to spread from one branch to the other, and this, as already explained, is an indication that the true centre of the disease is in the Gasserian ganglion itself, in which are located all the cell bodies of the sensory neurons which compose the fifth nerve. Surgeons should weigh carefully these facts when advising operation upon any isolated branch of any division of the trigeminus. That the disease tends to spread after operation from the affected branch to another is, however, no contraindication for the performance of the operation. A period of relief varying from a few months to as much as a year or two has often been gained for these sufferers, and this is a substantial gain well worth the cost and inconvenience of the operation. The tendency for the disease to spread from one branch to another is not increased, so far as we know, by operation on the affected nerve branch. The complete and satisfactory relief from suffering often secured by operation in cases in which only one nerve trunk is removed would seem to militate somewhat against the theory that the true seat of the disease is in the Gasserian ganglion, but too much reliance must not be placed upon this fact, because the powerful impression made upon an irritated sensory neuron, by cutting off a large portion of its axis cylinder or its dendrons, might be sufficient so to alter nutrition as to modify the ability of the neuron to generate or transmit painful impression. Physiology teaches that there is a distinction, important to the neuropathologist, between the irritability and the conductivity of a neuron or its axis cylinder, and that the former may be even abolished without the latter being impaired. (See section on Physiology of the Neuron.)

Again, as Donaldson has pointed out, the dendrons of the neurons are especially its excitable organs; in other words, they are adapted

especially for receiving impressions. Now, according to the observations of Lenhossek the fibres of all sensory neurons extending from the ganglion of the posterior root to their distributions in the skin are practically the true dendrons or the active sense organs of these sensory neurons. Hence in any operation on the peripheral branches of the fifth nerve the surgeon should recall that he is excising the most sensitive portion, *i.e.*, the receptive organs of the irritated neuron, and that therefore while he may give temporary relief to his patient he has no assurance that he is eradicating the whole of the diseased nervous tissue. If the seat of this disease is, as we suppose, in the cell body within the Gasserion ganglion, and if the disease has a tendency to pass from one group of these cell bodies to other neighboring groups, thus involving new nerve trunks, the operation will probably in most cases be followed by only temporary result. This in fact is what we know has happened after many of these operations on isolated trunks.

In cases in which the greatest intensity of pain seems to be located in one nerve trunk, but radiates as it were from that one with less intensity into others, an operation on that particular nerve will probably do no good. An instance of this is seen in the case in the Philadelphia Hospital quoted above. In that case a superficial operation on the superior maxillary nerve gave absolutely no relief.

Stretching the affected nerve has in some cases been attended with good results. J. F. Walsh [9] has recorded a case of severe tic douloureux limited to the lingual branch of the fifth nerve in a young woman aged twenty-six years. An incision was made on the side of the tongue and the nerve was exposed and dissected out of its bed. A weight of four pounds was applied for five minutes. The pain ceased entirely for three days but then reappeared. A few days later the nerve was again exposed and a weight of eight pounds was applied for two minutes, when it was reduced to four pounds which was continued for three minutes. The fibres of the nerves showed evidence of the tension. The pain now ceased entirely and at the end of seven years had not reappeared. Tension of this kind probably amounts practically to the same thing as excision, because the fibres of the nerve are probably disrupted by the weight.

In a case already referred to above, in which the pain was strictly limited for more than a year to the mental branch of the inferior dental nerve, a simple operation at the point of exit of this nerve from the mental foramen would probably have given complete but only temporary relief. Operation in this case was proposed, but was rejected by the patient and her husband. That the relief would only have been temporary was made evident from the fact that the pain eventu-

ally extended to the lingual and auriculotemporal branches of the third division of the fifth.

The only radical operation for severe tic douloureux involving several branches of the fifth nerve is undoubtedly complete removal of the Gasserian ganglion. This is a severe and heroic remedy, but considering the gravity of these cases it is perfectly justifiable if all its risks are fully explained to the patient and his friends. This is not the place to enter into an elaborate discussion of the technique of this grave operation. In a recent paper, W. W. Keen[91] has discussed elaborately this operation and has reported six cases in which he has operated. According to Keen the two methods of reaching the ganglion are, first, that devised by Rose and, second, the Hartley-Krause operation. He prefers the latter, first, because of its smaller mortality due to the possibility of more complete asepsis; secondly, because access to the ganglion is by a large opening; and, thirdly, because the entire ganglion with its roots can thus be removed. The operation consists of making an osteoplastic flap, shaped like a horseshoe, in the temporal region. This is turned down and the temporosphenoidal lobe is lifted from the middle fossa of the skull. This is done by the forefinger, the brain and its membranes being then held up by a broad spatula. The ganglion is thus reached and ablated. The dangers and difficulties of the operation consist especially, first, in hemorrhage from the middle meningeal artery and the vessels running in the dura mater, which requires to be torn away in order to expose the ganglion, and, secondly, in securing and removing the whole of the ganglion. In some cases, according to Keen, the ganglion is probably not entirely removed. It is simply broken up by the curette, hook, or other instruments. In one case, however, Keen succeeded in removing the ganglion entire. This distinction is a most important one, and the result, according to Keen, as regards recurrence or non-recurrence of the pain will probably depend upon the removal or non-removal of the whole of the ganglion. Among the results of the operation destruction of the cornea is especially to be feared. This, as already explained, is not an infrequent result of disease or injury to the fifth nerve. The first operation by Rose resulted in the loss of the eye. According to Keen, however, this grave result is not common after the operation. In one of his cases, however, a corneal ulcer resulted. Keen does not appear to regard danger to the eye as very great, but it cannot be denied that it exists: it may be due to drying of the cornea and to the presence of septic organisms in the eye. Keen recommends that the two lids be united in the centre, and that the eye be frequently inspected and if necessary cleansed.

The results in Keen's operations were as follows:

In Case I. the Gasserian ganglion was broken up after thirteen prior operations on various branches of the nerve. The patient was cured for twenty-six months, when he had a return of slight temporary twinges of pain.

In Case II. the Gasserian ganglion was broken up after eight prior operations on various branches of the nerve. Cure followed for eighteen months, when there was slight return of pain.

In Case III. the Gasserian ganglion was broken up after two prior operations on branches of the fifth nerve. Death resulted from septic meningitis.

In Case IV. the Gasserian ganglion was broken up after five prior operations on the nerve branches. Cure resulted for seven months. There was necrosis of the bone in the flap. There had been no return of pain at the time of the report.

In Case V. the Gasserian ganglion was broken up after two prior operations on the nerve branches. Cure resulted for two months so far as reported.

In Case VI. the Gasserian ganglion was removed entire with the second and third divisions and its sensory and motor roots back to the pons, after four prior operations on the nerve branches. Cure was complete, but the case was reported only a few weeks after operation.

These results, in spite of the fact that one death occurred in six cases, must be considered as highly satisfactory in such a grave affection as tic douloureux.

In a recent paper Doyen" recommends the resection of a portion of the bone covering the temporal fossa and of a part of the greater wing of the sphenoid bone. The technique of his operation need not be described here. The operation seems to render the complete removal of the ganglion rather more easy than the Hartley-Krause operation. In one case complete cure had persisted for two and a half years, but in two cases death resulted. Hence on the score of mortality the operation has much to be said against it.

From the strictly neurological standpoint removal of the Gasserian ganglion for intractable and widely extended tic douloureux is a logical and perfectly justifiable operation. This is so for the reason that all clinical evidence seems to point to the fact, as already emphasized in this paper, that the true seat of the disease is in this ganglion; consequently any operation upon an isolated trunk of the nerve will probably give only temporary relief. In cases in which the disease is as yet strictly limited to one nerve trunk, section of this trunk had of course better be undertaken instead of the dan-

gerous procedure for removing the ganglion itself. But in cases, unhappily the most frequent, in which more than one trunk is involved, nothing short of removal of the ganglion itself offers any prospect for a cure. With improved technique and enlarged experience the mortality from the operation, as seen in the work of Keen and other surgeons, is not so great as to discourage the removal of the ganglion.

Motor Affections of the Fifth Nerve.

The motor affections of the fifth nerve are of course confined to the motor branch of that nerve. This branch supplies the muscles of mastication, *i.e.*, the temporal, masseter, and pterygoid muscles, and some of the small muscles of the middle ear, as well as the mylohyoid muscle and the anterior belly of the digastric. Before dividing, however, into its several branches the motor division of the fifth nerve, after passing beneath the Gasserian ganglion, unites with a division of the inferior maxillary nerve, and from this trunk five branches arise which supply the muscles of mastication, as well as some of the integument about the mouth and the mucous membrane within the mouth beneath the buccinator muscle. Consequently these motor branches are mostly mixed nerves containing some sensory as well as motor filaments. The deep origin of the motor fibres of the fifth nerve is in a nucleus in the pons.

The motor division of the fifth nerve may be affected by lesions in its nucleus, in the pons, in its course between its exit from the pons and its junction with the inferior maxillary nerve, or in any of its branches. The most common affections are those that involve the nerve trunk in this second portion of its course, *i.e.*, between the pons and its junction with the branch from the inferior maxillary. The most common lesions are of course those that affect the sensory branch of the fifth nerve, as the two lie so close together that they are practically both affected by the same lesion. The most common of these lesions are syphilitic meningitis and tumors. Within the pons focal lesions, such as tumors, hemorrhage, and embolic softening, may cause paralysis of the motor root of the fifth nerve. The symptoms of paralysis of the motor division of the fifth nerve may be either spasm or loss of power.

Spasm of the muscles of mastication causes the well-known trismus or lockjaw. The jaw is firmly set and the muscles of mastication, especially the temporal and masseter, can be felt under the finger firmly contracted. Attempts to open the jaw meet with resistance, which may seem to be voluntary in character. When the

muscles of both sides are affected the face has the characteristic grin known as the risus sardonicus, caused by an elevation of the corners of the mouth due to a slight associated spasm of the facial muscles. Trismus as an isolated symptom is an exceedingly rare occurrence. As an associated symptom it is frequently seen in tetanus and in meningitis about the base of the brain. How much it is due in either of these cases to irritation of the nerve trunk, or how much to irritation of the nerve centres it is not possible to state. In meningitis about the base of the brain, especially the tuberculous variety, the motor branch of the fifth nerve may be within the inflamed membranes. In such cases the nerve branch itself may be directly involved and irritated by the morbid process. Reflex spasm in the muscles of mastication is said to be caused sometimes by irritation of some of the sensory branches of the fifth nerve. Such cases must be exceedingly rare. A carious tooth or an ulcer in the mouth may be capable of producing such a reflex spasm, but in any such instance I should rather regard the spasm as voluntary and indicative of an effort by the patient to render the painful part immobile and thus diminish all sources of irritation and all chances of increasing pain. Spasm of the muscles of mastication is an occasional symptom of tumor or other focal lesion in the pons or about the base of the brain. Thus Gowers refers to the case of a woman who probably had a focal syphilitic lesion about the basilar artery and who had spasm of the muscles of mastication. In this case, however, the sensory parts of the fifth nerve were unaffected, therefore it seems probable that the lesion could scarcely have been limited to the motor branch, but must have been nuclear. On the whole it must be concluded that spasm of the muscles supplied by the motor division of the fifth nerve is an extremely rare symptom of a local lesion within the cranium.

Paralysis of the motor branch of the fifth nerve is a usual accompaniment of paralysis of the sensory division. This can be accounted for by the close proximity of the motor nerve to the sensory root and the Gasserian ganglion. The symptoms are as follows: The temporal, masseter, and two pterygoid muscles on the affected side are paralyzed, consequently the patient is unable to close the jaws tightly except by the action of the muscles of the opposite side. This can readily be determined by causing the patient to chew upon some small object, such as a crust of bread or a piece of cork. With the fingers then placed over the temporal and masseter muscles it is readily observed that these do not contract, and this is in marked contrast with the motion of their fellows on the opposite side. The paralysis of the external pterygoid muscle causes a characteristic failure in the

lateral movements of the jaw. As the action of this muscle is to draw the lower jaw forward this movement cannot be satisfactorily executed, and the unopposed muscle acting alone the corresponding or unparalyzed side is not only drawn forward but deviates towards the paralyzed side; consequently the to-and-fro lateral movements of the lower jaw necessary for the trituration of the food are abolished, the patient in his effort simply forcing the lower jaw towards the paralyzed side. Although the tensor tympani is supplied by the fifth nerve, no affection of hearing is commonly caused by paralysis of the motor root. In long-standing cases, in which degeneration of the affected muscles occurs, a perceptible flattening is visible in the region of the temporal and masseter muscles, and secondary shortening may even produce a slight rigidity of the jaw on the affected side.

The diagnosis of paralysis of the motor branch of the fifth nerve is comparatively easy, although this paralysis may readily be overlooked by a careless observer. In any suspected case, as just remarked, it is only necessary for the physician to place the fingers of one hand upon the muscles of the affected side and the fingers of the other hand upon the muscles of the unaffected side and to direct the patient to chew upon some small object. The contrast between the action of the unparalyzed muscles and the loss of action of the paralyzed muscles is then very noticeable. The seat of the lesion is clearly indicated when the muscles are paralyzed in conjunction with anæsthesia of all the sensory branches of the fifth nerve. It must then be either within the pons or between the pons and the junction of the motor branch with the inferior maxillary nerve. The ophthalmic branch, after it leaves the Gasserian ganglion and enters the orbit, may of course be implicated without involving the motor branch. It is conceivable also that some branches of the second and third division of the fifth nerve might be implicated without involving this branch. The motor branch would not likely be involved apart from all the sensory branches except by a strictly localized lesion in its nucleus within the pons.

The *treatment* of paralysis of the motor branch of the fifth nerve depends of course upon its cause. In case of a syphilitic lesion active treatment with the iodides and with inunctions or hypodermic injections of mercury is indicated. Treatment of the affected muscles by electricity may be tried, but in case of a deep-seated lesion this treatment is of secondary importance. As the faradic contractility of the muscles is soon abolished by a destructive lesion it would be useless to try faradism. The galvanic current might be used to maintain the nutrition of the muscles during the progress of more radical treatment.

Diseases of the Seventh Nerve.

This nerve rises from a nucleus, in the tegmentum of the pons Varolii, composed of large multipolar cells. The axis cylinders from these neurons pass backwards underneath the floor of the fourth ventricle, where they turn forming the knee and then pass forward rather to the outer side of the nucleus and emerge from the pons close to its junction with the medulla oblongata, in close proximity with the eighth nerve. Within the bend or knee lies the nucleus of the sixth nerve.

The course of the facial nerve is a most important one for the neuropathologist. It first lies upon the crus cerebelli, but almost immediately, with the auditory nerve, enters the internal auditory meatus. It follows all the bends of the aqueduct of Fallopius through the petrous portion of the temporal bone. At its first and most acute bend

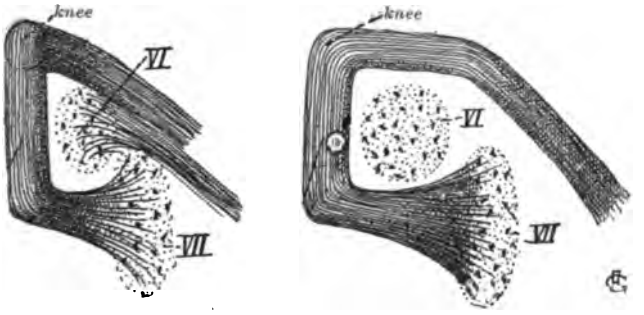

FIG. 22.—Diagram of Origin of the Seventh Nerve. On the left the fibres are seen in profile. VI., Nucleus of sixth nerve; VII., nucleus of seventh nerve. (Van Gehuchten.)

in this canal it forms the geniculate ganglion, at which point it is joined by the several petrosal nerves which connect the facial nerve with Meckel's ganglion, with the otic ganglion, and with the sympathetic system. The nerve then runs in the internal wall of the tympanum near the fenestra ovalis and emerges from the skull at the stylomastoid foramen. It now passes at once through the substance of the parotid gland near the external carotid artery and then divides into two main branches, which subdivide into the numerous branches which supply the facial muscles. The facial nerve supplies all the muscles of the face as well as the orbicularis palpebrarum and the frontal portion of the occipitofrontalis, the platysma, the buccinator, the posterior belly of the digastric, and the stylohyoid muscle. By way of the chorda tympani it sends branches to the lingualis. It also sends branches to the stapedius and the laxator and tensor tympani. It also probably supplies the levator palati and azygos uvulæ.

The important points to be remembered are as follows: Within

the aqueduct it sends off the tympanic branch, which supplies the stapedius and laxator tympani muscles, and the chorda tympani nerve which leaves it about a quarter of an inch before its exit (Gray, Van Gehuchten[23]). It is important to recall that the chorda tympani is not purely a branch of the facial nerve, but conveys sensory fibres as well: it is probably in fact a gustatory nerve for the anterior portion of the tongue. The trunk of the facial nerve within the aqueduct lies in the internal wall of the tympanum, and consequently it may be affected by destructive lesions of the middle ear. These relations supply data for the localization of the disease processes which affect the trunk of the nerve. Sapolini believes that the chorda tympani is simply a continuation of the intermediary nerve of Wrisberg; that the trunk of this nerve becomes united to but not identified with the trunk of the facial, and that the geniculate ganglion in the aqueduct of Fallopius is analogous to the ganglia which exist on all the sensory roots of the cerebrospinal system. His and Martin have found in this geniculate ganglion in human and cat embryos bipolar cells identical with those that are found in the cerebrospinal ganglia of all embryos; and Retzius has described in this same ganglion in the adult cat, dog, and man unipolar cells identical with those found in the cerebrospinal ganglia in all adult mammals (Van Gehuchten). According to this we have a thirteenth cranial nerve which may be regarded as the sensory mate of the seventh nerve.

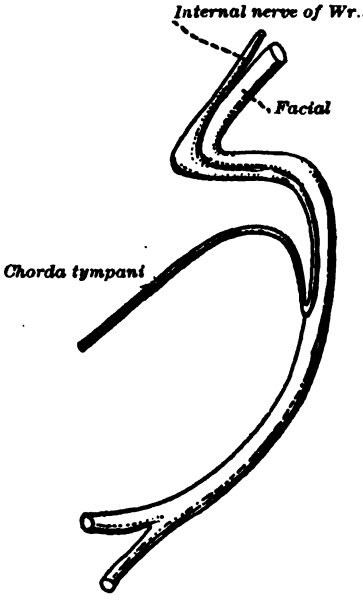

FIG. 23.—The Thirteenth Cranial Nerve of Sapolini. (Van Gehuchten.)

The most common disease of the seventh nerve is neuritis at its emergence from the stylomastoid foramen. This inflammation may not always be confined to the trunk after it leaves the skull, but in some cases probably extends for some distance up into the aqueduct of Fallopius. As, however, in the majority of these cases there is no evidence of involvement of the chorda tympani (which, as we have seen, leaves the trunk of the facial nerve only a quarter of an inch within the aqueduct), it is not probable that the inflammation often

extends in these simple cases of neuritis far into the bony canal. The chief cause of this neuritis of the facial nerve is exposure to cold. In many cases a direct history can be obtained from the patients of such exposure. Thus, sitting in a draught when overheated, or sitting at an open window of a railway car or in an open vehicle on a cold day, are among the common causes of the affection. Another less common cause is disease of the petrous portion of the temporal bone. Such disease may be caused by suppurative processes within the middle ear. The trunk of the facial nerve, as will be recalled, lies in the wall of the tympanum separated by only a thin layer of bone from the cavity of the middle ear. The carious process may very readily break down this thin wall of bone. Thus in a case of a man suffering from middle-ear disease local treatment by an aural surgeon was followed in a short time by inflammation and paralysis of the facial nerve. Such a case is peculiarly liable to reflect unjustly upon the skill of the aurist. In any case of chronic otitis such a mischance may occur without operative interference. It has even been claimed that inflammation may extend to the trunk of the facial nerve from the middle ear without destruction of this thin lamina of the bone.

Syphilitic disease of the temporal bone or about the base of the skull may also involve the seventh nerve.

Injury of various kinds may also cause a paralysis of the facial nerve. Thus surgical operations on or about the parotid gland may involve the trunk of the nerve. Blows upon the cheek in the neighborhood of the nerve may cause facial paralysis. In obstetrical practice it is important to recall that pressure of the forceps has caused such a paralysis in the child. Fracture of the base of the skull may also involve the facial nerve, although such cases are rare.

Lesions of various kinds about the base of the brain may cause paralysis of the facial nerve. Such especially are tumors, meningitis, and hemorrhage.

Within the pons various small focal lesions may injure the nucleus or the extensive intrapontine fibres of the seventh nerve. Thus a small hemorrhage or neoplasm may paralyze this nerve. In case the fibres of the facial nerve are involved near the knee the nucleus of the sixth nerve may also be involved, thus giving a combined paralysis of the sixth and seventh nerves. Paralysis of the seventh nerve has also been caused in disseminated sclerosis by a small islet of degeneration, and in locomotor ataxia also the nerve has, although very rarely, been involved.

Symptoms of paralysis of the facial nerve are quite characteristic. When the nerve trunk is affected at any point so as to involve all its

cleus or in its root above the point of connection with the nerve of Wrisberg taste is not affected; so also when the nerve is injured by blows on the cheek or by a neuritis limited to the course of the nerve after it leaves the aqueduct of Fallopius taste is not affected. In some cases, however, of inflammation at or about the stylomastoid foramen the chorda tympani may be involved by the extension of the inflammation a short distance within the Fallopian canal to the origin of this branch. As a consequence involvement of the sense of taste in facial paralysis is of considerable localizing importance. It shows that the chorda tympani is involved anywhere from the point of union of the intermediary nerve of Wrisberg with the seventh nerve up to the point of branching of the chorda tympani. The anterior half only of the tongue on the affected side is involved in this loss of taste.

In some cases of facial paralysis the stapedius muscle is paralyzed and as a result there may be a slight tinnitus. As a rule, however, hearing is not impaired in pure facial paralysis, and when dulness of hearing does occur seemingly in connection with these cases it is probably due to involvement of the middle ear in an inflammatory process.

The paralysis caused by destructive lesions of the seventh nerve is of the purely peripheral type. This is so in whatever part of its course the nerve is involved, from its nucleus in the pons to its distribution in the facial muscles. The paralysis, therefore, is not only complete but of the flaccid type. The muscles lose their tone and their reflex excitability. They degenerate, atrophy, and what is very characteristic soon show the reactions of degeneration (see page 74). These reactions of degeneration in facial paralysis are usually very prompt to appear, and as they are exhibited in such convenient groups of superficial muscles they may be studied in these cases with great advantage. The faradic contractility usually decreases rapidly and disappears in the course of a few days. The changes to galvanism as a rule are also prompt. First there is the initial increase in galvanic excitability, then there are the gradual decrease and the qualitative changes. Thus, the cathodal closure contraction decreases while the anodal closure contraction equals or exceeds it. In extreme cases the cathodal opening contraction may be elicited. The modal change also occurs. This consists, as already explained, in a change in the manner or mode of reaction of the muscular tissue to the galvanic current. Instead of the quick, sharp response of a normal muscle the response is slow and sluggish, and there may even be a continuation of the contraction while the current continues to flow. This latter is called duration tetany.

The reasons for the phenomena of degeneration and flaccid paraly-

sis in all cases of destructive lesions of the seventh nerve in or below its nucleus are not difficult to understand. As already explained in the preceding part of this article, the nutrition and functional activity of the axis cylinder and of the muscles which it supplies depend upon the integrity of the cell body of the neuron. If any portion of an axis cylinder is cut off from its nutritive centre, *i.e.*, the cell body, degeneration of the axis cylinder as well as paralysis and degeneration of the muscles which it supplies will result. In the case of the seventh nerve the cell body of the peripheral neuron is located in the nucleus in the pons, hence any lesion in this nucleus or in the course of the nerve fibres anywhere between this nucleus and their distribution in the muscles will cause the phenomena of flaccid paralysis and degeneration. The only variation that will occur according to the location of the lesion in paralysis of the seventh nerve will depend upon what strands of the nerves are involved or what contiguous structures are implicated. In all cases the type of a flaccid paralysis with degeneration involving practically all the muscles will remain. In case of nuclear lesion there may of course be symptoms caused by destruction of other important pontine structures. Thus there may be hemiplegia due to involvement of the motor strands passing through the pons. There may be involvement of the sixth nerve if the lesion is in the neighborhood of the knee of the seventh nerve. In basilar lesions the sixth or the eighth nerve or even the fifth may also be involved, while in lesions at various points within the aqueduct of Fallopius the branches of the seventh nerve, such as those which go to structures within the tympanum or the chorda tympani, may be involved.

The distinction between a peripheral palsy of the seventh nerve just described (*i.e.*, the palsy due to any lesion of or below the nucleus of the nerve in the pons) and a paralysis of the facial muscles due to a pure central lesion (*i.e.*, a lesion above the nucleus anywhere as far upwards as the motor cortex) will be easily understood from the description just given. The first important point in the peripheral lesion is that all the facial muscles, including the orbicularis palpebrarum and the frontalis, are involved, while in central lesions or lesions above the nucleus anywhere as far up as the motor cortex only the lower half of the face is involved in the paralysis, the orbicularis palpebrarum and the frontalis being especially conspicuous by their exemption. A second point of distinction is that the type of paralysis in a peripheral lesion, *i.e.*, a lesion of or below the nucleus, is flaccid and accompanied, for reasons already stated, with the reactions of degeneration, while in central lesions (*i.e.*, all lesions above the nucleus) the type of paralysis is not flaccid. There is no

wasting or degeneration and the reactions of degeneration are never seen.

It is important to bear in mind, however, that in many cases of peripheral facial palsy the extreme type of reaction of degeneration already described is not always seen. There may be only a partial reaction of degeneration. In such cases the faradic contractility may be merely diminished, while the serial changes do not present a typical formula. These facts are due to the nerve in some instances being the seat of only a mild grade of inflammation, or to some fibres being more involved than others.

Although the facial nerve is purely motor, yet some symptoms of pain at the point of exit of the nerve upon the cheek may be present in the early stages. There may for instance be pain on pressure or a sense of soreness about the face and cheek. In cases in which giddiness is present this is probably due to an extension of the inflammatory process to the tympanum, and is not due to involvement of the facial nerve *per se*. Swelling of the cheek is very rare, and if present is probably due to some infiltration of the tissues surrounding the nerve trunk.

The *course* of facial paralysis varies with the cause. In even the milder cases, however, the affection as a rule is not evanescent. In all cases due to neuritis the onset of the disease is rather sudden. In a few hours or a day at most the disease is firmly established. This somewhat sudden appearance of facial or Bell's palsy is the cause sometimes of much mental disquiet or even alarm to the patient. He is apt to think that the paralysis is a symptom of grave, deep-seated lesion in the brain. Even mild cases may and usually do persist for weeks. A guarded prognosis is always advisable in every case, because at the onset it is impossible to foretell whether the course of the disease will or will not be slow or even whether a permanent paralysis may result. Among the most important indications for prognosis are the completeness of the paralysis and the early establishment of the reactions of degeneration. In any case, for instance, in which all the muscles supplied by the facial nerve after its exit from the stylomastoid foramen are paralyzed completely and in the course of a few hours or a day, and in which the faradic contractility is promptly abolished and the reactions of degeneration are early secured, the probability is that the case will be a protracted one. The prognosis on the whole is of course more favorable in a simple neuritis due to exposure to cold than in those cases in which the paralysis is due to a deep-seated destructive lesion such as caries in the temporal bone. Still, cases of neuritis, especially of the kind just referred to, are sometimes exceedingly obstinate and may even be followed by

permanent paralysis and contracture of the affected muscle. I have known a simple uncomplicated case of Bell's palsy persist for three or four months and then be followed by complete recovery. In young persons I think the prognosis on the whole is rather more favorable than in middle age and in the senile.

In cases in which paralysis of the facial nerve is due to carious processes in the temporal bone (such, for instance, as are caused by disease of the middle ear), the prognosis should be even more guarded than in cases of simple neuritis. Such cases as a rule are obstinate and rather more apt in my observation to lead to permanent loss of power and deformity of the face. This can be readily understood when it is reflected that the damage to the nerve in such cases amounts truly to a trauma as well as to an inflammation. The nerve trunk in such cases is injured and compressed by the crumbling walls of its canal and is held firmly in a narrow space. It is consequently inflamed and compressed to a more injurious extent than occurs on the exterior of the skull. Moreover, the disease process is a much more actively virulent and infectious one than is that caused by exposure to cold. Consequently it is not uncommon for the nerve to be irreparably damaged in these cases, and even when this is not so the injury may be so great as to lead to a long and tedious course before recovery is attained.

In cases in which paralysis of the seventh nerve is due to syphilitic meningitis at the base of the brain the prognosis, in spite of the hope held out by specific treatment, should be guarded. In such cases it is not uncommon for the eighth nerve to be involved with the seventh, with consequent complete deafness and other symptoms such as severe headache and possibly even epileptic seizures. In one such case seen by me in the Philadelphia Hospital the woman had complete paralysis of the seventh and eighth nerves with occasional vertigo, vomiting, and epileptiform attacks, and persistent headache. She had a distinct history of syphilis, but the case was exceedingly rebellious to specific medication and remained unchanged for many months.

In all cases in which paralysis of the seventh nerve is caused by organic disease about the base of the brain or in the pons, the prognosis is unfavorable. In such cases the seventh nerve shares the fate of the other portions of the nervous system that are involved. In cases of tumors no amelioration as a rule can be expected. Even when some particular symptom remits or intermits nothing practically is gained because the course is really progressive.

In cases of fracture of the skull or other injury involving the nerve trunk, in its course either within or without the skull, the prognosis

will naturally depend much upon the severity and extent of the injury. No rule can be laid down that will cover all such cases. As in the case of neuritis, however, the general rule will hold good that the sooner and more completely the paralysis of all the facial muscles is established, and the more promptly typical reactions of degeneration occur, the more unfavorable is the prognosis. These reactions of degeneration, as already explained, indicate the degeneration of the axis cylinder, which is caused only by its complete severance from its nutritive centre, the cell body of the neuron. From what has been said, it follows that the duration of facial palsy varies according to its cause. In mild cases of Bell's palsy due to neuritis recovery may occur in a month or six weeks, although such cases in my observation are very rare. In the more severe cases in which the reactions of degeneration are complete the duration of the case may be so long as four or six months. The same may be said of injuries to the facial nerve in which degeneration is once complete.

In those who do not recover, the paralysis which is left may improve to some slight extent. This improvement in some cases is only apparent, and is due to the fact that slight secondary contraction occurs in the paralyzed muscles and tends to restore the symmetry of the face. In long-standing cases, in fact, this secondary contraction may even draw the face slightly over towards the paralyzed side. In many of these hopeless cases the orbicularis palpebrarum seems to regain a little power so that the patient can partially close and protect the eye. Some of this protection is apparently gained by the patient permitting the upper eyelid slightly to droop and thus to shade the eye. Some of it is also no doubt due to the slight secondary contraction of the paralyzed muscle.

Even in those who satisfactorily recover so that practically all functions of the affected muscle are restored there may still remain for a long while a slight paresis distinguishable by an expert observer.

The pathology of ordinary Bell's palsy is now well understood, although it has been in the past a topic of some controversy. In acute neuritis the process is probably always confined to the main nerve trunks composing the *pes anserinus*, and from them extends upwards to or into the Fallopian canal. Whether or no this inflammation is ever a true peripheral neuritis—*i.e.*, an inflammation starting in the peripheral nerve endings within the muscles and thence spreading upwards with diminishing intensity towards the nerve centre—is perhaps an open question. There seems to be no adequate reason for denying that such a distinct peripheral neuritis might occur in the seventh nerve just as it occurs in the nerves of the extremities. We know for instance that this is a common type of inflammation in

the nerves of the arms and legs in cases of alcoholic neuritis. But alcoholic neuritis is very rare in the seventh nerve. In most cases of multiple neuritis in topers the seventh nerve is not involved, while, on the other hand, in the vast majority of cases of pure and simple Bell's palsy the question of alcohol does not arise as an etiological factor. Hence the cases of Bell's palsy are hardly analogous to the cases of alcoholic multiple neuritis. In the former the action of alcohol is not conspicuous, and in the latter paralysis of the seventh nerve is not commonly seen. Therefore there is no reason to suppose that in the great majority of cases of Bell's palsy the inflammation is truly peripheral in seat. It is more probably, as already said, an inflammation of the main trunk of the nerve with its main branches. This inflammation is a diffused interstitial inflammation, such as has already been described in a previous section (see page 51).

The inflammatory process probably extends in most cases some distance into the Fallopian canal, but there is no reason for the dogmatic assertion that it always extends as far as the geniculate ganglion. On the contrary, in many cases it probably does not extend even as high as the point where the chorda tympani leaves the main body of the nerve. This is proved by the fact that in many cases of Bell's palsy there is no paralysis of the sense of taste.

In cases due to caries of the petrous portion of the temporal bone the inflammation of the nerve is probably of a higher grade of intensity and more destructive than in the cases due to cold.

In cases accompanying syphilitic meningitis the nerve trunk is probably the seat of true syphilitic or gummatous infiltration. In destructive lesions such as tumor the process in the nerve is probably one of compression and consequent destruction of the axis cylinders.

The *treatment* of Bell's palsy should be prompt and thorough. It should be begun with a blister as large as a silver dollar applied over the point of exit of the nerve. In the case of a man the beard of course should be shaved over a sufficient area to admit of this blister, but when the beard is habitually worn it is doubtful whether the cheek should be entirely and cleanly shaven; the protection which the beard affords in such cases is probably beneficial and the patient need not be exposed to any slight risk that arises from shaving it off. After the blister has drawn, warm fomentations should be applied, and it is well in beardless individuals especially that the face should be protected for a period of several weeks by some covering so as to avoid any further injurious effects of exposure to cold. I believe that these cases are sufficiently important to demand complete rest and seclusion in the house for a week or more, and such a course of treatment will probably tend to shorten the duration of

the disease. Internal medication is probably of little avail in these cases. Two drugs especially (salicylate of sodium and iodide of potassium) are almost universally recommended, but I have not been able to assure myself that they are of much benefit. If used, the salicylate of sodium should be given in full doses, as much as ten or fifteen grains being given three or four times a day. The iodide of potassium, in doses of five or ten grains three times a day, may be given during the continuance of the paralysis or until the symptoms of iodism appear.

The best remedy for inflammation of the seventh nerve is undoubtedly electricity. This, however, should be applied with caution at first. In fact during the very early stages of the case, *i.e.*, during the first week or two, electricity had probably better not be used. The choice of the current depends very much upon the severity of the symptoms. In cases in which the faradic contractility is early abolished it is quite useless to apply the faradic current. In mild cases, however, in which the muscles still respond to it, the faradic current may be used in a mild strength. In severe cases, in which the reactions of degeneration are fully established, treatment should be commenced at once with the galvanic current. This should be used by applying one small sponge electrode over the affected muscle while a large flat sponge electrode is held on the back of the neck or on the sternum. The muscles should be gently stroked, sufficient strength being gradually turned on to cause an active but not too violent contraction. This treatment may be applied for about ten minutes daily. If too strong currents are used it is possible that injury may be done, and in long-standing cases that even secondary or permanent contraction of the muscles might be hastened.

In obstinate cases gentle massage of the affected muscle may be used. Dana recommends, in cases in which the corner of the mouth tends to droop and the unopposed muscles of the sound side are unduly vigorous, that the corner of the mouth should be drawn up by means of a bent hook which is fastened behind the ear. The object of this is to take off the strain and restore tone to the paralyzed muscles.

In cases in which irritation or inflammation of the conjunctiva results, care should be taken to cleanse this with antiseptic solutions and to protect the eye with a shade.

In cases in which recovery is slow hypodermic injections of strychnine may be used in the affected muscles, but very great care should be observed that the syringe is thoroughly aseptic so that no septic abscesses or points of induration are caused.

Diseases of the Eighth Nerve.

The eighth or acoustic nerve is the nerve of hearing. As it is exclusively a sensitive nerve we should expect to find that it has its real origin outside of the cerebrospinal axis, according to the plan of the nervous system already described. The eighth nerve is divided into two main branches or rather is composed from two main stems, viz., the vestibular and the cochlear nerves. These two unite in the bottom of the auditory meatus to form the main trunk of the auditory nerve, which then passes up and into the central nervous system. I speak of the auditory nerve as passing *into* the nervous system instead of *out* of it, since it has its true origin outside of the cerebrospinal axis.

According to Van Gehuchten there is found upon each of the two

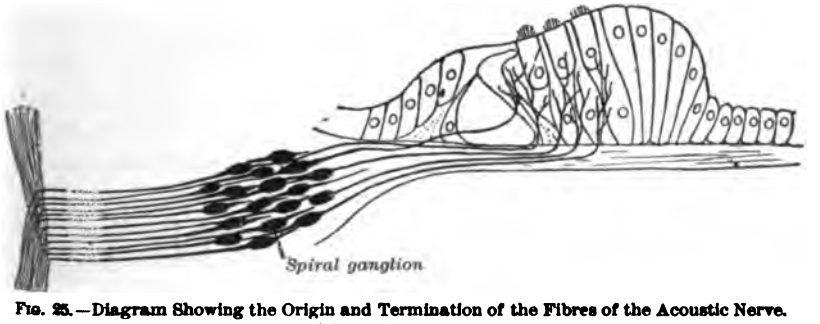

FIG. 25.—Diagram Showing the Origin and Termination of the Fibres of the Acoustic Nerve. (Van Gehuchten.)

main stems or branches of the auditory nerve a mass of nerve cells which constitutes a ganglion. The cochlear branch presents at the base of the spiral lamina a ganglion called the spiral ganglion or the ganglion of Corti. The ganglion of the vestibular branch of the acoustic nerve is situated at the bottom of the internal auditory meatus. It is known as the vestibular ganglion or ganglion of Scarpa. The researches of Retzius, Van Gehuchten, Cajal, and Lenhossek have demonstrated that these ganglia are composed of bipolar cells and that the peripheral prolongations of these cells (dendrons) terminate by free ramifications; that the ramifications of the cells of the spiral ganglion are distributed to the epithelium of the organ of Corti, and of the ganglion of Scarpa to the epithelial cells of the maculæ acusticæ in the labyrinth. The prolongations brainward of these bipolar cells are the true axis cylinders and form the trunk proper of the acoustic nerve. When the nerve reaches the

metencephalon it divides into two branches, called anatomically roots. The external or cochlear root passes to the lateral tubercle and its fibres enter the accessory nucleus, terminating in the gray substance. The internal or vestibular root enters between the inferior cerebellar peduncle and the descending root of the fifth nerve. Its fibres enter the nucleus of Deiters, some ultimately reaching the cerebellum.

The diseases of the eighth nerve may be divided into those that are caused by the abolition of function and those that depend upon irritation.

The abolition of the function of the eighth nerve may depend upon a variety of causes. Some of these causes act upon the nerve proper and some upon its accessory or peripheral organ. Among the most common causes of complete nerve deafness is meningitis at the base of the brain, causing inflammation and compression of the nerve trunk. Syphilis may cause such a meningitis. In such a case other nerves, especially the seventh, may be involved and the patient may present other symptoms of brain syphilis. In one patient (already referred to in this article) complete paralysis of both the seventh and eighth nerves coexisted with intense headache, vomiting, and a few epileptic attacks. The patient had a history of syphilis and the lesion was probably a syphilitic meningitis about the roots of the seventh and eighth nerves. Tumors at the base of the brain and in the cerebellum, pons, and medulla oblongata may cause complete abolition of the function of the auditory nerve. According to Mills and Lloyd [11] diminution or loss of hearing, as well as tinnitus and hyperæsthesia of hearing, is occasionally observed. These symptoms are most likely to be manifested by tumors of the base of the brain or of the cerebellum, in such a position as to involve the auditory nerve or auditory tracts. According to these authors tinnitus, with complete or partial deafness, accompanied with facial paralysis, with or without paresis of the limbs of the opposite side, indicates clearly a tumor of the base of the brain so situated as to involve the superficial origin or intracranial course of these nerves. This statement, however, needs to be modified to the extent at least that such symptoms may be produced, as already said, by basilar meningitis.

Cerebrospinal meningitis may cause deafness by pressure or involvement of the auditory nerve. In those who recover from this disease the deafness is sometimes permanent. According to Stillé,[11a] vertigo, as originally described by Miner in 1823, may occur from the very commencement of the attack. In some epidemics, as that of Strasburg, this vertigo is so great as to interfere with walking and

to produce forced or whirling movements. In all such instances the auditory symptoms are no doubt caused by inflammation or pressure upon the delicate structure of the auditory nerve. According to Strümpell purulent inflammation of the auditory nerve in cerebro-spinal meningitis may be propagated as far as the labyrinth or even into the middle ear. Auditory symptoms, especially deafness, often occur during this fever, and according to Burnett "the deafness is very frequently permanent. In cases of very young children this may not be discovered until convalescence. This deafness is often profound and in both ears. A peculiar affection of the gait, the so-called sailors' gait, may result. Burnett thinks that the lesion is a neuritis descendens, *i.e.*, a slow encroachment of the inflammation upon the labyrinth along the auditory nerve. Moos, quoted by Burnett, found that in sixty-four cases of deafness following cerebro-spinal meningitis fifty per cent. had disturbance of equilibration and hearing; fifty-nine per cent. were totally deaf in both ears and hence became deaf-mutes; thirty-one and a half per cent. were totally deaf, but retained speech, while only one and a half per cent. recovered. Moos found that those patients who retained the sense of hearing for high notes, even when there was absolute deafness for low notes, had a better prognosis for retaining hearing for spoken words. It is thus seen that involvement of the auditory nerve in cerebro-spinal meningitis is a most grave affection and that permanent deafness is a not infrequent result. The prognosis is especially unfavorable if absolute deafness persists for more than three months.

The poison of mumps sometimes causes deafness. Burnett thinks that this occurs by true metastasis, that the labyrinth and the nerve structures of the inner ear are involved in inflammation. The early symptoms are tinnitus, vertigo, and deafness, but the tinnitus and vertigo may disappear, leaving only the deafness. Disturbances of equilibration may or may not occur, and the symptoms may not develop until after the other metastatic processes have subsided. Vomiting and nausea, as well as alteration in gait, suggest the labyrinthine seat of this affection. Pain also may be a symptom. Instead of inflammation the lesion may be a serous exudation into the labyrinth. Deafness fortunately is a rare sequel of mumps.

Other causes of deafness, acting upon the internal ear, are the excessive use of quinine and salicylic acid, injuries, hemorrhage, syphilitic disease, and, in rare instances, neoplasms. Permanent deafness is not infrequently caused in persons who are constantly exposed to loud noises, as, for instance, boiler-makers and locomotive engineers. This influence, however, is probably somewhat exaggerated. In an inquiry which I formerly made in the Southwark

Machine Works in Philadelphia it appeared that deafness was not a very common symptom in the boiler-makers in that establishment. Finally a primary atrophy of the auditory nerve, just as of the optic nerve, occurs very rarely in locomotor ataxia. I have seen it in one instance only. In this patient, a man aged about fifty, with well-marked locomotor ataxia, extreme deafness was present and was evidently due to an involvement of the nerve.

The chief symptom of complete abolition of the function of the eighth nerve is deafness, but this may be accompanied at some stage with tinnitus and vertigo. These symptoms of irritation may appear early in the course of the affection and may lessen or even disappear as the function of the nerve is entirely abolished. In some cases, however, tinnitus may be a persistent symptom, but it probably always indicates that the function of the nerve is not entirely abolished. In some diseases deafness comes on rather suddenly. This is characteristic of syphilitic disease of the internal ear (Dana[9]). In cases in which the nerve is involved in a basilar meningitis other nerves also, as already explained, may be implicated. Thus the seventh nerve may be involved and numerous other symptoms, such as headache and even hemiplegia due to the wide spread of the infectious process, may be observed. In cases in which the function of the nerve is entirely abolished bone conduction is lost; thus a tuning fork held upon the bones of the skull is not heard through the affected nerve. In such cases aerial conduction is also lost, although some authors state that it may be partially preserved. In case it is preserved, however, the function of the nerve certainly cannot be entirely abolished.

Diseases of the auditory nerve or of its end organ, the ear, that cause symptoms of irritation are well recognized. As a rule diseases of the auditory nuclei within the pons do not cause tinnitus, but all irritative lesions of the nerve trunk or of the internal ear may cause both tinnitus and vertigo. Thus meningitis, such as has already been described as a cause of deafness, may cause also symptoms of irritation. Blows and other injuries to the head, tumors about the base of the brain, sunstroke, chronic alcoholism, and excessive use of tobacco may cause these symptoms. The arterial degeneration of advanced life sometimes causes giddiness and ringing in the ears, but the exact mechanism of the symptoms under these circumstances is not plain. The most common cause of combined deafness, tinnitus, and vertigo is organic disease of the internal ear, especially of the labyrinth. Such diseases are inflammation and degeneration, extending especially from the middle ear, and in some instances probably minute hemorrhages. In all such instances of involvement of the in-

ternal ear the structure of the auditory nerve is probably ultimately affected. Degeneration and consequent abolition or perversion of function result.

The symptoms of irritative lesions of the inner ear and of the auditory nerve are tinnitus, vertigo, and various degrees of deafness.

The *tinnitus* of ear disease assumes a great variety of forms. In the majority of cases it is a sighing or whistling sound or the sound of escaping steam. These sounds vary in pitch and intensity according to circumstances. They even vary in individual cases, sometimes being much louder and more disturbing than at others. These variations probably depend upon variations in general health or in the intensity of the disease process or even in some cases upon atmospheric conditions. Sometimes the tinnitus is of a louder, rumbling character. There may be paroxysms of great intensity, during which loud throbbing or roaring sounds are heard. These sometimes usher in attacks of vertigo or a period of increased deafness. In some cases the tinnitus is of a throbbing or beating character, and may even be synchronous with the heart beats; but this is not always an indication that the tinnitus is dependent, as some claim, upon a lesion of the vascular system, such as aneurysm. It is more probably due to implication of the small blood-vessels or capillaries in the disease process within the internal ear. Alterations in the blood states, such as anæmia or plethora, may be factors in causing this symptom. Tinnitus as a rule is a simple, not an elaborate sound; in other words, it does not assume distinct forms such as tunes or words. In cases in which these occur there is reason to suppose that the affection depends upon some brain lesion, especially in the cortex. Exceptions to this rule seem to occur in cases in which patients mistake the tinnitus for more elaborate sounds. Thus in falling asleep patients with tinnitus occasionally imagine they hear distant music or the voices of persons talking. In the insane it is even possible that tinnitus may be the starting-point of auditory hallucinations. Persons who are perfectly sane, however, and even in their waking moments, may occasionally mistake tinnitus for the sound of voices or other external objects.

The location of the morbid sound varies somewhat. In the great majority of cases it is in the ear. In some, however, it is located in other parts of the head; thus it may be deep seated as though it were far within the recesses of the brain. In some it is even in the occiput or in the vertex. It may be in one or both ears, according as these are or are not both involved, or it may be much louder in one ear than in the other.

The intensity of the tinnitus may vary according to external cir-

cumstances. Thus in noisy places it may be quite imperceptible. It is usually worse in the stillness of the night, and then may become so accentuated as seriously to interfere with the patient's repose. The explanation for these facts is probably to be found not in the variation in intensity of the tinnitus itself, but only in the variation in external noises. When these latter are great the sound of the tinnitus is drowned or obscured; but when they are absent entirely the tinnitus is perceived with increased intensity, simply because the attention is not distracted from it.

Tinnitus is always a disturbing and in some cases a very serious symptom. It may even be so great as entirely to dominate the patient's mental life. It distracts the attention, harasses the patience, and destroys the capacity of the sufferer for the enjoyment of life. It is thus apt to cause profound neurasthenic, hypochondriacal, or even melancholy states. The patient falls into a condition of self-absorption, irritability, or mental depression, and the general health may even suffer seriously.

Vertigo is another symptom of disease of the auditory nerve or perhaps, more properly speaking, of the internal ear, especially the labyrinth. It is usually a paroxysmal or intermitting symptom and is always as a rule associated with tinnitus or deafness or both. It is considered by aurists as primarily due to an involvement of the labyrinth, hence its name, labyrinthine vertigo. It is caused especially by diseases that directly affect the labyrinth, such as congestion, inflammation, and hemorrhage, or by those that indirectly affect it, such as otitis media, thickening of the ear drum with ankylosis of the ossicles, obstruction of the Eustachian tube; or by pressure on the ear drum by wax or any foreign body in the external ear. Vertigo varies greatly in frequency and intensity as a symptom of ear disease. It is by no means necessarily commensurate with the intensity of either the tinnitus or the deafness. In some cases of persistent tinnitus and of progressive deafness from disease of the middle ear vertigo occurs only at very long intervals, in fact a patient may have not more than four or five attacks in as many years. These isolated attacks, however, may be of great severity, although of short duration. In other cases, vertigo is much more common. It may occur at intervals of a few days, and the patient may be conscious of a slight tendency to it during the periods of comparative freedom. In some of these cases too the individual attack may be of long duration, forcing the patient to keep in a supine position. In cases in which the vertigo is paroxysmal, each attack may be ushered in by prodromal symptoms. These are usually alterations in the character and intensity of the tinnitus. Thus a patient with a mild grade of tinnitus of a sigh-

ing or whistling character may suddenly have loud rumbling noises in the ear. These are quite alarming, especially as the patient soon comes to know their import. They last for but a few moments as a rule, and as they subside the vertigo comes on. These paroxysms of vertigo sometimes come without apparent exciting cause. At other times, however, a distinct cause for them may exist. Sudden changes in attitude, for example, especially suddenly raising or lowering the head, may cause an onset of severe vertigo with the prodromal symptoms just described. Thus a gentleman with slight tinnitus and progressive deafness, worse in one ear than the other, experienced a paroxysm of most intense and alarming vertigo on suddenly raising his head after stooping to pick up an object on the floor; this was ushered in by loud rumbling sounds, which abated before the vertiginous symptom appeared. The paroxysm, which was confined to the better ear, seemed to usher in a somewhat rapid increase of deafness in that ear.

The vertiginous sensation is variously described. It is commonly said to consist of a feeling as of surrounding objects whirling around, or of the patient's body being whirled around, or of both. This is the usual mode of description, although the truth is aural vertigo is literally neither one nor the other of these. It is in fact a sensation *sui generis* and is not precisely like an ordinary attack of giddiness such as may be artificially induced by whirling the body around many times. It is a distressing affection of the organ or sense of equilibration quite impossible to be described by one who has not felt it. During its continuance the patient is usually quite helpless; he clutches for support some neighboring object, or holds his head in his hands. As a rule he at first keeps the head erect, although later, if the symptom persists, he may seek to lie down. He experiences great alarm and usually cries out and asks for aid. In very severe cases the patient reels and may even fall violently to the ground. This, however, is not in the nature of a forced movement, but due entirely to the loss of power of equilibration. Muscular spasms or fits do not occur in these attacks of vertigo. The movements made by the patient are either wildly voluntary in an endeavor to save or support himself or they are the results of weakness and incoördination due to the loss of equilibrium. In very severe cases the duration of the paroxysm, even though short, seems unending to the sufferer. It leaves an impression on the mind for a long while of dejection and of apprehension of a recurrence of similar attacks.

Accompanying the vertigo there is usually pallor of the face and some sweating; the eyes have a startled and staring expression at first with perhaps slight oscillatory movements, but later the patient closes

them as though to shut out the sight of surrounding objects. Accompanying or following the attack nausea and vomiting may appear, although this is by no means constant. In paroxysms of great severity there may be no nausea whatever.

The patient does not lose consciousness in aural vertigo. He may be in part oblivious to his surroundings and even to the inquiries of friends, but this is due entirely to the distress which he is suffering and of which he is perfectly conscious. As the attack passes off the patient's color returns and the tinnitus, from which he is usually a chronic sufferer, resumes its customary note. A slight increase in deafness may be noted for a few days, perhaps may even be permanent. This deafness may be more marked to some musical notes than to others. In some cases the vertiginous symptom does not promptly abate; although the paroxysm in its great intensity passes by, there may persist for some days a slight giddy sensation. This may be so great in some few instances that the patient is obliged to keep his bed, any attempt to assume a sitting or standing position increasing the vertigo.

The *prognosis* of aural vertigo varies according to circumstances. Even in cases of progressive deafness and persistent tinnitus the vertigo, as already said, may be a rare and infrequent symptom. I have known of a case in which an interval of more than four years has passed after a very severe paroxysm without the recurrence of another. There seems to be no rule that applies to these cases. The non-recurrence of the vertigo is by no means a sign that the tinnitus and deafness will not continue. In cases, however, in which the vertigo is a frequent symptom the prognosis so far as hearing is concerned is poor. Such cases usually progress to a stage of great deafness. In cases in which the labyrinth is only secondarily involved, as for instance in middle-ear disease and in cases of impacted cerumen in the external ear, removal or amelioration of the cause may greatly benefit this symptom.

The *diagnosis* of aural vertigo must be made specially from vertigo caused by affections of the eye, the brain, the stomach, and in certain general states, as, for instance, gout, neurasthenia, plethora, and possibly anæmia. The distinction is best made by the absence or presence of associated symptoms. Aural vertigo is inevitably associated sooner or later with tinnitus and deafness. These do not occur as a rule in any other condition, except in the vertigo due to organic disease of the brain, especially when this involves the auditory nerve or its associated tracts in the brain. The vertigo of ocular defects is usually caused by insufficiency of the internal recti muscles. This is commonly met with in myopia (Mackenzie). In gastric

vertigo there is usually some chronic gastric disorder or even organic disease. There may be pain or a sense of uneasiness after eating, especially if the diet has been inappropriate. Heartburn, eructations, and a sense of distention in the stomach and chest are usually experienced. In the vertigo of gout or of neurasthenia, anæmia, etc., the diagnosis is readily made from the associated symptoms. In all these conditions anything that tends to aggravate the general constitutional state may cause slight vertiginous attacks. These may be accompanied even with slight buzzing in the ear, but persistent tinnitus and deafness are unknown. Excessive use of tobacco, alcohol, tea, or coffee, may possibly tend to cause this symptom in such patients. So also will the vitiated atmosphere of crowded rooms. In the vertigo of organic brain disease other symptoms, such as headache, paralysis, convulsions, and affections of the mental faculties will usually clear up the diagnosis. Vertigo is an occasional symptom of epilepsy, but in this disease it is usually associated with loss of consciousness and convulsions.

The third and last prominent symptom of irritative and destructive lesions of the auditory nerve or its end organ is deafness. This is usually progressive. In very rare instances only does it occur suddenly. Thus in injuries to or hemorrhage in the internal ear deafness may be a sudden symptom, but in the great majority of instances it comes on gradually. Its approach in fact is sometimes so insidious that the patient may for a long time not observe it. He may even have some vertiginous attacks which he is apt to attribute to some disorder of digestion, and he may experience a slight tinnitus to which at first he attaches no importance. As these symptoms of irritation proceed, however, he becomes conscious finally of impairment of his hearing, usually at first only in one ear. The deafness in all these cases is probably coincident with the onset of the tinnitus, and the vertigo may be a signal symptom of danger, the true significance of which is not recognized. It is not probable in any case of a beginning irritative lesion of the ear or the auditory nerve that there is any initial hyperacuity of hearing. The deafness is the result of increasing impairment of the conducting power either of the nerve or of its end organ, the irritative symptoms proper being the tinnitus and vertigo.

Deafness varies in its characteristics in different persons and in the same person at different times. As a rule hearing for low notes is abolished first. High and clear notes are often readily recognized by persons comparatively quite deaf. It is not the loudness of the note so much as its character that is essential for hearing.

In long-standing cases in which the affection is limited at first to

the middle and internal ear the conducting power of the nerve itself usually becomes impaired. This may be due merely to disuse, but is more probably a symptom of a true degenerative process in the nerve fibres.

The three symptoms already described, *i.e.*, vertigo, tinnitus, and deafness, constitute, when combined, what is known as Ménière's disease. This was first described by this observer in 1861. Its essential, as it was described by Ménière, is supposed to be an affection of the labyrinth, although in most of these cases other regions of the internal ear, as the semicircular canals and cochlea, are probably involved; as is made evident by the associated tinnitus and deafness. It is doubtful, in fact, whether Ménière's disease can be properly looked on as a distinct morbid entity. Diseases of the middle and internal ear exist in all grades of severity, and these various symptoms, tinnitus, vertigo, and deafness, vary according to the extent of the disease and the exact structures involved. The term "Ménière's disease" seems to be restricted by otologists to that severe grade of aural disease in which these three symptoms are more or less constantly associated, and in which especially the vertigo assumes a paroxysmal and inveterate type. Cases of increasing deafness with tinnitus are very commonly observed in which vertigo is never seen or is at most but rarely experienced. In such cases we may suppose that the labyrinth is not seriously involved, and by common consent these cases are not included under the term, Ménière's disease.

The *treatment* of Ménière's disease and allied affections of the middle and internal ear is largely relegated to the aural surgeon. During the paroxysm of vertigo little if any treatment as a rule is required. The attack, even when most severe, is often of short duration and passes off without treatment. In those cases, however, in which the vertigo persists, the patient should be kept strictly in a recumbent position. He sometimes assumes by preference a position on his side or face rather than on the back, or he may even in rare cases prefer to sit up holding his head in his hands. No arbitrary rule governing his position is perhaps needed or desirable. Bromide of potassium in large doses, twenty to thirty grains, should be administered. Charcot recommended heroic doses of quinine, as much as ten grains, three times a day. This drug in such doses seems to be given on the theory that it deadens the sensibility of the auditory nerve, and that in grave or hopeless cases its destructive action on the nerve, leading to permanent abolition of function, *i.e.*, permanent deafness, might be preferrable to a continuation of the intolerable vertigo. Gelsemium and salicylate of sodium have been

recommended, but their action cannot be relied upon. Counter-irritation over the mastoid region, such as by a blister or even by the actual cautery, have, according to Mackenzie, proved serviceable. Recently Burnett and others have recommended excision of one or other of the ossicles of the ear in the hope of relieving tinnitus and vertigo, but the utility of this operation seems still to be a subject of discussion. In many cases of middle-ear disease proper treatment of the throat and posterior nasal chamber and of the internal ear is useful. Massage of the ear drum, when this is sclerosed and retracted, and when, as is probably frequently the case, the small bones of the middle ear are sclerosed, is a useful procedure. Strawbridge, of Philadelphia, has devised a small electrical engine for this purpose. It acts by causing rapid vibration of the ear drum.

In diseases of the auditory nerve proper, such as those caused by meningitis or organic disease at the base of the brain, little can be done by way of medication. In syphilitic cases, however, the patient should be given the benefit of full and persistent antisyphilitic treatment. This is best attained by heroic doses of the iodide of potassium, and in recent cases by the hypodermic use of some appropriate mercurial preparation. It may be proper to state here that in all cases of recent syphilitic affections of the nervous system a prompt action of mercurials is probably much better obtained by the hypodermic use of mercury than by any other method. At the recent Congress of French Dermatologists and Syphilographers at Lyons it was claimed that when mercury is given in pill form the metal in some cases could not be detected in the urine at all. When given by inunction it did not appear in the urine for a week or ten days, but when given hypodermically it could be found sometimes within twenty-four hours. These facts seem to prove that the mercurial salt is much more rapidly absorbed and hence exerts its influence much more forcibly when thrown at once into the circulation by being injected under the skin. The reason for this probably depends upon the well-known fact that syphilis acts upon the nervous system by way of the coats of the blood-vessels and the capillaries. Hence when introduced directly into the circulation it exerts all its power directly, promptly, and energetically upon the primary seat of the lesion.

Diseases of the Ninth Nerve.

The ninth or glossopharyngeal nerve is the sensori-motor nerve of the base of the tongue and of the pharynx. The motor fibres of this nerve arise from large cells in the superior portion of the nucleus ambiguus in the medulla oblongata. Its nucleus of origin is close to that of the pneumogastric nerve. The superficial origin of the glossopharyngeal nerve is immediately behind the olivary body, in close relationship with the trunk of the tenth or pneumogastric nerve. Soon after leaving the medulla the nerve presents two small ganglia, the superior and petrous ganglia. As in the case of all sensory nerves, the peripheral sensory neurons of the glossopharyngeal nerve have their cells of origin outside of the cerebrospinal axis, and these cells are located especially in this petrous ganglion, which corresponds exactly to the ganglia on the posterior roots of the spinal nerves. According to Van Gehuchten, these ganglia have the same structure as those of the spinal nerves. They are formed of unipolar cells, the single branch of which soon bifurcates and gives origin to a peripheral and a central branch. The peripheral branch terminates in the mucous membrane of the pharynx and tongue. The central branch becomes the axis cylinder, penetrating into the medulla at the upper part of its posterior surface, and like all sensory neurons divides into an ascending and a descending branch. The descending branch enters the solitary fasciculus or descending root of this nerve, and can be traced for some distance into the middle of the bulb, where it terminates by collateral and terminal ramifications in the neighboring gray matter. The ascending branch is shorter; it constitutes rather a horizontal branch and terminates directly in the upper part of a nucleus of gray matter beneath the floor of the fourth ventricle, which Van Gehuchten describes as the trigone of the glossopharyngeal and vagus nerves. This mass of course does not constitute a nucleus of origin, but a terminal nucleus for the sensitive fibres of these two nerves.

The superficial origin of the ninth nerve is by five or six roots arising from the sulcus between the inferior cerebellar peduncle and the olive, just above the superficial origin of the strands of the vagus. The filaments of origin of the ninth nerve soon unite in one trunk. This pierces the dura mater, from which it receives a sheath. Some of its posterior filaments present two ganglia, named respectively, as already said, the petrous, and the jugular or superior ganglia. The nerve leaves the skull by the jugular foramen in close proximity to the pneumogastric and spinal accessory nerve. After its exit from

the skull it is in close proximity to the jugular vein and the internal carotid artery. It passes close along the border of the stylopharyngeus muscle. It is finally distributed to the mucous membrane of the fauces, the base of the tongue, and the posterior part of the mouth and the tonsils. The glossopharyngeal nerve has communicating branches by way of the petrous ganglion, with, first, the pneumogastric, second, the superior cervical ganglion of the sympathetic, and third, the facial nerve just as it emerges from the stylomastoid foramen. The petrous ganglion also sends a branch, which gives sensibility to the internal wall of the middle ear. This is called the tympanic nerve or nerve of Jacobson. This nerve passes by a minute canal—the tympanic canal—into the cavity of the tympanum, where it is distributed to the mucous membrane of the tympanum and the Eustachian canal. The other collateral branches of the glossopharyngeal nerve are the pharyngeal to the lateral wall of the pharynx, the stylopharyngeal which innervates the muscle of the same name, and the tonsillary branches distributed to the mucous membrane of the tonsils and of the pillars and vault of the palate.

The terminal branches of the glossopharyngeal nerve are distributed especially to the mucous membrane of the posterior third of the dorsum of the tongue and to the neighborhood of the epiglottis. The lingual branches of the nerve give to the mucous membrane of the posterior surface of the tongue its general sensibility, and also supply filaments of special sensibility for the sense of taste.

According to Van Gehuchten, the observations of Fusari and Panasci have seemed to demonstrate that the gustatory fibres of the glossopharyngeal nerve have their cells of origin in the mucous membrane of the tongue itself. These authors have described the presence in the taste bulbs of bipolar cells identical with those in the olfactory mucous membrane of the nose. The peripheral branches of these cells terminate freely upon the surface of the mucous membrane, while the central branch becomes a fibre of the nerve itself. Van Gehuchten himself, however, as well as Retzius, Lenhossek, and others, whom he quotes, has not been able to demonstrate the presence of these cells in the taste bulbs. He therefore holds to the view that the gustatory fibres of the glossopharyngeal nerve have their cells of origin in the two ganglia called the superior and petrous ganglia, already described, on the trunk of the nerve in the neighborhood of the jugular foramen. It is interesting to note, however, that the claim of Fusari and Panasci (that the cells of origin of the fibres of taste are situated in the mucous membrane of the tongue) makes the fibres strictly analogous to the fibres of the olfactory nerve; and that in this respect they seem to occupy a position analogous to the per-

ipheral sensory neurons in the earthworm which Lenhossek has demonstrated in the epithelium of the skin.

The functions of the glossopharyngeal nerve are largely sensory. It supplies the posterior part of the tongue, the regions of the palate and tonsils, and the pharynx with common sensation. It also appears to supply the posterior part of the tongue with the special sense of taste. It has also some motor fibres which supply the glossopharyngeal muscle. Its precise functions, both sensory and motor, are still the subject of considerable discussion among anatomists and physiologists. That the nerve contains motor fibres seems indubitable from the fact that its nucleus of origin in the upper part of the medulla oblongata presents cells of a distinctly motor type. These cells evidently supply motor fibres to the stylopharyngeus muscle. The sensory functions of the nerve so far as common sensibility is concerned are not disputed. The nerve undoubtedly is the nerve of common sensation of the regions specified. In reference, however, to the sense of taste, there still seems to be uncertainty in the minds of some. As we have just seen, Fusari and Panasci have claimed that they have demonstrated the presence of the cell body of the neuron of taste in the taste bulb of the tongue. The central branches of these neurons, according to the same observers, pass brainward by the trunk of the glossopharyngeal. Other observers, however, notably Van Gehuchten, Retzius, and Lenhossek, have not been able to determine the presence of these peripheral gustatory neurons. It seems probable that we shall have to wait for more careful research according to the new methods, before we can determine positively the physiology and anatomy of taste. The older writers indulged largely in speculation, and sought to trace the gustatory fibres without sufficient warrant in the most roundabout routes, by way of the various connections of the seventh, ninth, and fifth nerves. So far as I can judge, these speculations have little scientific warrant or value. Clinical experience proves that the anterior portion of the tongue is supplied with the sense of taste by the chorda tympani nerve. The posterior part of the tongue, however, as already said, is supplied apparently with gustatory fibres by the ninth or glossopharyngeal nerve. On the other hand the fifth nerve is thought by some to conduct taste fibres, and there is clinical evidence for this. The difficulties of estimating not only the functions but also the diseases of this nerve are greater perhaps than in the case of any one of the twelve cranial nerves. It is an extremely difficult nerve, for instance, for physiologists to experiment upon in the lower animals, while in the human subject it is scarcely if ever the seat of isolated disease. I know of no instance, in fact, in which it can be claimed without doubt or criti-

cism that the nerve trunk has been diseased alone. The facts being thus, it seems that we must either fall back on mere speculation as to what disease might or should do with regard to taste, or else acknowledge our relative ignorance on these questions.

The glossopharyngeal nerve may, however, be affected in some disease processes. In that degenerative disease, for instance, known as glosso-labio-pharyngeal paralysis, the nucleus of the ninth nerve is sometimes involved in the degenerative process. Here, however, the symptoms caused by an involvement of this nerve are so obscured by the symptoms caused by involvement of other nerves that it is difficult to separate them. No doubt, however, some of the anæsthesia of the fauces and impairment of the sense of taste as well possibly as some of the paralytic symptoms in the act of swallowing are caused by degeneration of the nucleus and roots of the glossopharyngeal nerve.

The trunk of the glossopharyngeal nerve might, of course, be involved in focal lesions of the medulla oblongata or in localized lesions about the base of the brain, such as tumors, meningitis, and hemorrhage. Surprisingly little, however, has been observed of symptoms of paralysis of this nerve by such lesions. Possibly this may be due to the fact that all symptoms produced by lesions of the glossopharyngeal nerve are deep seated and readily overlooked unless searched for with the most careful tests. A patient, for instance, might readily have anæsthesia of one side of the pharynx and loss of taste on one side of the posterior third of the tongue, without this being very perceptible to himself or very readily detected by his physician. It is doubtful whether the unilateral lesion would seriously interfere with the act of swallowing. This act being largely reflex, no doubt depends to some extent upon the integrity of the sensory fibres in the glossopharyngeal nerve, but the irritability might readily be blunted, we may suppose, or even abolished on one side without the act of swallowing being more than slightly embarrassed. It is impossible to say, owing to lack of observation, to what extent speech, especially the formation of gutturals, might be impaired by complete paralysis of the glossopharyngeal nerve. It is probable that this nerve conveys the muscular sense from parts of the pharynx and fauces, and the abolition of this we might suppose would cause some incoördination of the movements necessary for speech; but of this we have no positive clinical evidence. As the nerve supplies common sensation to and about the epiglottis, its paralysis would tend to cause choking by permitting small particles of food to find their way into the windpipe.

According to Mills and Lloyd,[*] taste may be involved in various ways by brain tumors. Thus subjective sensations of taste, particu-

larly the so-called metallic taste, may be present in cases of brain tumor. This is probably caused by irritation of the trunk of the glossopharyngeal nerve, especially of its petrous ganglion or of its connections within the medulla. As these authors suggest, the abnormal taste phenomena observed in rare cases of brain tumor may be caused by irritation of the nerve trunk in the same way that a mild galvanic current applied to the nape of the neck will often cause a metallic taste.

As the glossopharyngeal nerve apparently supplies common sensibility to the tympanum by way of the nerve of Jacobson, its paralysis of course would cause anæsthesia of the cavity, provided the lesions were in or above the petrous ganglion; but this symptom would be extremely difficult to determine.

The motor symptoms caused by paralysis of the glossopharyngeal nerve, due to a strictly localized lesion of the nerve itself, are also extremely difficult to estimate. The nerve seems to supply the stylopharyngeal muscle, but the isolated action of this muscle is probably not important and its isolated paralysis would not cause conspicuous symptoms. It is possible that other of the pharyngeal muscles are also supplied by the glossopharyngeal nerve, but anatomists differ on this subject. Its pharyngeal branches help to constitute the pharyngeal plexus which supplies the muscular fibres of the upper part of the pharynx. Some claim, however, that the fibres which supply these muscles are derived from the pneumogastric. As an isolated paralysis of the glossopharyngeal nerve is extremely rare, it is impossible to determine this point definitely.

The *treatment* of diseases of the glossopharyngeal nerve resolves itself usually into treatment of lesions involving this nerve conjointly with others. In labio-glosso-pharyngeal paralysis the treatment is usually unsatisfactory. Strychnine and electricity are probably the most reliable agents, but the disease is usually steadily progressive and exceedingly rebellious to medication. In cases of tumor, meningitis, or other gross lesions involving this nerve the treatment is that which has been indicated elsewhere for these lesions, but it is usually most unsatisfactory.

Diseases of the Tenth Nerve.

The tenth nerve, called also the pneumogastric or vagus nerve, has an extensive distribution. It is composed of sensory and motor fibres and supplies organs and viscera in the neck, chest, and abdomen. It arises from the medulla oblongata in the sulcus just behind the olivary body and below the fibres of origin of the ninth nerve.

As it is composed of both motor and sensory fibres, it of course has both a motor and a sensory nucleus. Its motor nucleus is situated deep in the medulla or postoblongata and is known as the nucleus ambiguus. It is practically the same nucleus from which arise motor fibres of the ninth nerve. This nucleus contains large cells, evidently of the motor type, which are the cell bodies of the peripheral motor neurons of these two nerves. From these cell bodies arise axis cylinders, which after a slightly bent course pass out from the medulla in the sulcus behind the olivary body. The nucleus ambiguus, from which these motor fibres arise, is practically a continuation upwards of the gray matter constituting either the anterior or the lateral horn of the spinal cord. Hence both these nerves may be looked upon as approaching the type of the spinal nerve.

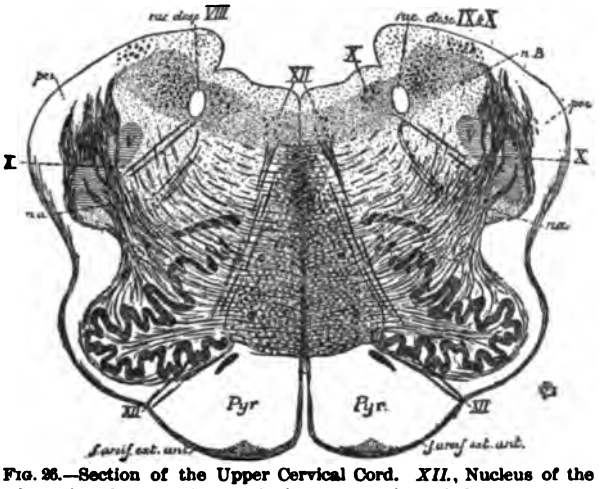

FIG. 26.—Section of the Upper Cervical Cord. *XII.*, Nucleus of the hypoglossal nerve; *X.*, terminal sensory nucleus of the pneumogastric nerve; *nB*, Burdach's nucleus; *pci*, inferior cerebellar peduncle; *na*, nucleus ambiguus. (Van Gehuchten.)

The superficial origin of the pneumogastric nerve is by about twelve roots issuing from the medulla in the lateral fissure behind the olivary body. These roots coalesce to form the trunk of the nerve.

The sensory fibres of the pneumogastric have their origin outside of the cerebrospinal axis. The cell bodies of these peripheral neurons are placed in ganglia called the jugular and the plexiform ganglia. The former is situated nearer to the central origin of the nerve. These are analogous to the ganglia of the posterior roots of the spinal nerves. According to Van Gehuchten, they are formed of unipolar cells, the single stem of which divides into a central and a peripheral branch. The peripheral branch passes by way of the nerve trunk to terminate in the mucous membrane to which the nerve is distributed. The central branch becomes the axis cylinder and passes into the medulla oblongata by way of the roots of the nerve. Within the medulla it divides into a descending and ascending branch. The descending branch becomes a part of the solitary fasciculus, while the ascending or horizontal branch terminates in the inferior portion of a gray mass

under the floor of the fourth ventricle called the "trigone" of the vagus and the glossopharyngeal nerves (Van Gehuchten). This gray mass has been considered for a long time as the true nucleus of origin of the pneumogastric nerve. According to the new methods of staining, however, and especially in the light of the embryological work of His, we know now that this gray mass constitutes a *terminal* nucleus. The axis cylinders of the peripheral sensory neurons of both the ninth and tenth nerves terminate by their arborizations in this gray mass, and in it are located the nerve cells of the sensory neurons of the second order whose axis cylinders penetrate farther into the cerebro-spinal axis.

The course and distribution of the pneumogastric nerve are extensive and complex. At the point of formation of its trunk from its numerous small roots of origin there is found the jugular ganglion and at a short distance lower down its trunk expands into a second or plexiform ganglion. The significance of these has already been stated. These ganglia represent the ganglia on the posterior roots of the spinal nerves, although in the case of the pneumogastric nerve they are not located on a separate sensory root, but are incorporated in the main trunk of the nerve. This nerve trunk passes through the dura mater and emerges from the cranium in company with the ninth nerve by the jugular foramen. It descends vertically through the neck just in front of the vertebral column, being enclosed within the sheath of the great vessels of the neck. After it passes into the thoracic cavity the anatomical relations differ on the two sides. On the right side it passes between the subclavian artery and vein and gives origin at the inferior border of this artery to its recurrent branch, or right inferior laryngeal nerve. The main trunk of the pneumogastric passes behind the right bronchus. On the left side the trunk of the nerve crosses the arch of the aorta and at this point gives origin to the inferior or recurrent laryngeal nerve, which winds around the aorta and then ascends to the side of the trachea (Gray).

The branches of the pneumogastric nerve may be stated in brief as follows: In the jugular fossa the auricular branch arises, which apparently supplies the integument of the back part of the pinna.

In the neck the pneumogastric gives origin to the pharyngeal, superior laryngeal, recurrent laryngeal, and cervical cardiac branches. The pharyngeal branch anastomoses with branches from the glossopharyngeal and sympathetic to form the pharyngeal plexus, which supplies the muscles and the mucous membrane of the pharynx with motor and sensory fibres. The superior laryngeal nerve arises from the plexiform ganglion of the pneumogastric and is apparently largely a sensory nerve for the larynx. The inferior or recurrent laryngeal is the motor nerve for the larynx. The cervical cardiac

branches of the pneumogastric form connections with the sympathetic and with the cardiac plexus and cardiac nerves.

The thoracic cardiac branches terminate in the deep cardiac plexus.

Within the thorax the branches of the pneumogastric nerve are the anterior and posterior pulmonary and the œsophageal. The former two enter into the formation of the pulmonary plexus and have important connections with the sympathetic system. The œsophageal branches go to form the œsophageal plexus.

The trunk of the pneumogastric nerve penetrates the diaphragm and distributes its terminal filaments by way of the gastric branches. These gastric branches are distributed principally to the stomach and the cœliac and splenic plexuses. They form connections also with the sympathetic system and with the hepatic plexus (Gray, Van Gehuchten).

The functions of the pneumogastric nerve have been the object of much investigation and some speculation. The nerve is concerned in the acts of deglutition, phonation, circulation, respiration, digestion, and possibly even in some of the functions of the intestines. Taking in order its main branches from above downwards we may briefly map out its physiology.

Its first important branch is the pharyngeal. This arises from the plexiform ganglion on the trunk of the nerve and passes to the lateral wall of the pharynx where it anastomoses with branches of the glossopharyngeal and sympathetic nerves to form the pharyngeal plexus. From this plexus are supplied the branches of innervation for the muscles and mucous membrane of the pharynx. According to this plan it seems that the pneumogastric enters only in part into the innervation of the pharyngeal muscles and mucous membrane. Exactly what part of these it supplies is still a question. According to Rethi, quoted by Van Gehuchten, the pharyngeal filaments of the vagus nerve innervate the constrictor muscles of the pharynx as well as some of those of the vault of the palate. The nerve is evidently important in relation to deglutition. It not only supplies some of the pharyngeal muscles, as already said, but probably also controls the movements of the œsophagus and even of the cardiac orifice of the stomach.

The next important branch of the pneumogastric nerve is the superior laryngeal. It has two branches. The first supplies the cricothyroid muscle as well as the lower constrictor. The other branch supplies the mucous membrane of the larynx, the vocal cords, the epiglottis, and the base of the tongue. Thus it is largely the sensory nerve of the larynx, although, as is seen, it has some motor fibres. It is probably largely upon this nerve that the fine adjustments of deglutition and respiration depend. In the act of

swallowing it is necessary of course that both the sensory and motor supply should be in strict harmony in order that the epiglottis may close properly and that no particles of food should drop into the larynx. These functions depend probably in great part upon the pneumogastric nerve.

The recurrent branch of the pneumogastric nerve is the motor nerve of the larynx. It supplies all its muscles with the exception of those already mentioned. It also sends branches to the pharynx, trachea, and œsophagus. This branch is evidently largely motor, although it is possible that it supplies part of the mucous membrane of the pharynx with sensory fibres. It is proper to state that the motor innervation of the larynx has been a much disputed question. Some have claimed that this is a function of the pneumogastric nerve, others of the spinal accessory. Grabower" claimed that the spinal accessory nerve has nothing to do with the innervation of the larynx and that the vagus is the only motor nerve of this organ. He has recently recorded a case of tabes in which there was degeneration of the left recurrent nerve and of the extrabulbar portion of the roots of both tenth nerves with perfect integrity of the roots of both eleventh nerves, degeneration of the roots of both ninth nerves, integrity of the nuclei and intra-bulbar portion of the roots of the tenth and eleventh nerves, and moderate degeneration of both spinal roots of the fifth nerve. The patient had bulbar symptoms and as can be seen the recurrent nerve was degenerated as well as the roots of both tenth nerves, while the roots of both eleventh nerves were intact. This, according to Grabower, proves that the accessory has nothing to do with the innervation of the larynx. He claims, however, to know of sixteen cases in which paralysis of the larynx was observed in connection with paralysis of the trapezius; seven of these were of peripheral origin and therefore, he claims, useless for the question, because a peripheral disease in the sheaths of the blood-vessels of the neck would likely involve both the pneumogastric and spinal accessory nerves. In the other case there was no autopsy. Oppenheim agrees with Grabower that his case demonstrates that the vagus and not the accessorius is the motor nerve of the larynx. It is possible, he thinks, that the tabetic virus affects the roots of the vagus and not the nuclei, as is seen in cases of some other poisons.

The cervical cardiac branches go to form the cardiac plexus. One of these fillets, springing directly from the trunk of the vagus or formed by the union of a branch of the superior laryngeal nerve with a branch from the trunk of the vagus, constitutes the nerve known as the depressor (not inhibitor) of the heart, or the nerve of Cyon. It has long been known that the pneumograstic nerve exercises a pow-

erful inhibitory action on the heart. It controls the heart movements, and its irritation, especially the irritation of its inhibitory branch, retards the beating of the heart. Under this influence the heart beats more slowly. A strong electric current, it is said, will stop the heart in diastole.

The thoracic cardiac branches of the pneumogastric also go to form the cardiac plexus, through which the action of this nerve is exercised upon the heart.

The anterior and posterior pulmonary branches form with the sympathetic the anterior and posterior pulmonary plexus respectively. From these two plexuses arise numerous branches which follow the bronchial tubes through all their subdivisions into the interior of the lungs. This nerve thus evidently has an important function in regulating respiration. It probably conducts afferent branches through the pulmonary plexus from the structure of the lungs and bronchi. These afferent or sensory impressions probably lead to reflex acts of breathing. In respect to respiration, however, it must be recalled that it is a complex movement depending upon the proper adjustment of the mouth, nose, pharynx, and larynx. It thus requires an extensive coördination of muscular movements, guided and in a sense controlled by numerous afferent or sensory impressions. This extensive complex movement is, therefore, not dependent upon any one nerve, but upon a coördinate or harmonious action of several nerves, especially the fifth or trifacial, the seventh or facial, the ninth or glossopharyngeal, the tenth or pneumogastric, the eleventh or spinal accessory, the spinal thoracic nerves, and the sympathetic fibres. All of these nerves, it is to be remembered, have numerous anastomosing branches. In addition, the branches of the pneumogastric nerve, distributed to the bronchi, cause constriction of these tubes, as seen in asthma. According to some observers the laryngeal nerves, both superior and inferior, exert an inhibitory action upon the movements of respiration (Habershon[*]). It is supposed by some that the afferent impulses carried from the lungs to the respiratory centre in the brain by way of the pneumogastric nerve depend partly upon the accumulation of carbonic acid in the blood. This excites the reflex action of respiration.

The œsophageal branches of the pneumogastric nerve descend upon the anterior and posterior surfaces of the œsophagus, sending out large numbers of anastomosing branches which constitute the œsophageal plexus. From this plexus arise fibres which supply the muscles and mucous membrane of the œsophagus. The function of these nerves is evidently to preside over the reflex action of the œsophagus in the process of swallowing.

In the abdomen the terminal branches of the pneumogastric nerve are supplied to the muscles and mucous membrane of the walls of the stomach and extend by way of the hepatic plexus also to the liver. The nerve sends branches also to the semilunar ganglion and to the solar plexus. An important function of the pneumogastric nerve is indicated by these distributions. It is probable that the proper performance of digestion depends in a measure upon this nerve. The movements of the œsophagus and stomach are under its control. As the nerve also contains sensory fibres, it is probably the path by which we are acquainted with both the normal and the morbid sensations in that organ. For instance, it possibly conveys the sensation of nausea and consequently is active in the function of vomiting. Its action upon the liver is somewhat obscure. The well-known experiment of Claude Bernard illustrated its function. He discovered that puncture of the floor of the fourth ventricle at the trigonum of the pneumogastric nerve produced saccharine urine, probably as a result of a disturbance of the glycogenic function of the liver. The vagus finally may have some connection through the sympathetic system with the suprarenal capsule, the kidney, and even with the uterus. This connection may possibly explain the reflex irritation of the stomach caused by diseases of these organs.

To recapitulate, we may refer to the experiments made by physiologists by division of the pneumogastric nerve. According to Habershon division of the pneumogastric nerve causes loss of sensation in the throat and larynx so that foreign substances may pass unheeded into this organ; there is loss of power in the throat and of action of the vocal cords, and the glottis is partially closed; the heart's action is impeded, but at first its frequency and force are increased; the lungs lose their sensibility; the movements of the chest wall are less active, and respiration becomes less frequent and more deep; the capillaries of the lungs are filled with blood, hence the mucous membrane is congested; trophic changes occur and inflammation in the lung tissue is induced; in the stomach the cardiac orifice does not act; the mucous membrane becomes pale and secretion at first is checked. Irritation of these nerves causes vomiting and movements of the stomach, but complete division of them stops vomiting and the stomach is paralyzed and may become distended.

Isolated disease of the pneumogastric nerve is exceedingly rare. On the other hand, the nerve is not infrequently affected by diseases of neighboring parts and organs. Distinct inflammation of the pneumogastric nerve, in the sense in which the facial nerve, for instance, is inflamed, is practically unheard of; so too, its deep course through the neck and thorax renders it almost exempt from injury.

Its nucleus in the medulla may be involved in the degenerative process which also attacks nuclei of other cranial nerves, producing such combined scleroses as the labio-glosso-pharyngeal paralysis. In such a case the disease process is essentially a destructive one, falling primarily upon the cell body of the neuron rather than upon its axis cylinder. Hemorrhages, tumors, and meningitis about the base of the brain might involve the trunk of the pneumogastric nerve, but in all such instances there would be many other complicating symptoms. Tumors in the neck and very rarely injuries might also involve the trunk of the nerve. Because of its deep course, however, such injuries are rare, and if they occurred they would probably involve also the large blood-vessels of the neck and would be speedily fatal. In the thorax the commonest cause for involvement of the pneumogastric nerve or more especially of its recurrent laryngeal branch are aneurysms of the aorta and its branches, and tumors in the anterior mediastinal space. Diseases of the lungs, even the most extensive and destructive in character, do not, as a rule, impair the action of the pneumogastric nerve. Gastric disorders may possibly act as irritants to this nerve; in fact some of the symptoms of gastric irritation are probably conveyed to consciousness by way of the pneumogastric. The various metallic and other poisons do not seem, as a rule, to have much influence upon the pneumogastric nerve. In lead poisoning, however, severe præcordial pain is sometimes present and may possibly be an expression of irritation of some of the fibres of this nerve. In grave alcoholic poisoning we also see a symptom occasionally that suggests the possibility of paralysis of the cardiac branch of the nerve known as the inhibitor of the heart or nerve of Cyon. Thus in acute alcoholic multiple neuritis there is present sometimes an obstinate and even dangerous tachycardia. This is extremely rebellious to treatment and will not respond for instance to digitalis. It is sometimes the cause of sudden death in this disease. Thus in one instance in a woman with all the symptoms of peripheral alcoholic neuritis the heart beat persistently in spite of rest in bed and full nourishment at one hundred and forty pulsations to the minute. Sudden death occurred in this case from the application of an ice bag over the chest. The so-called "tobacco heart" is possibly an affection in part of the pneumogastric nerve (see below). Syphilis as a rule does not play an important rôle in affections of the pneumogastric. A syphilitic inflammation of this nerve trunk has not occurred so far as I know, although the nerve has been involved in syphilitic meningitis. Exposure to cold never acts as an exciting cause of disease of the pneumogastric nerve.

This nerve is supposed to be variously influenced or involved in

some of the obscure so-called gastric and other neuroses. In migraine, for instance, it is probably involved. This disease, as already explained, seems to expend its force especially in the territory of the optic, trigeminus, and pneumogastric nerves. Thus amblyopia, headache, and vomiting are its three chief characteristics. What the exact affection of the nerve is, however, in migraine, it is impossible to say. If this disease, as some suppose, is due to a toxin in the blood, we can only surmise that this poison acts in some way as an irritant upon the trunk or the nerve endings of the gastric branches of the pneumogastric. But why it singles out these three nerves especially is an unsolved problem in pathology. There are other gastric, abdominal, and cardiac neuroses that apparently involve the pneumogastric nerve, but the subject is so obscure that it is scarcely worth while to speculate about it. In many of these cases the patients are either neurasthenic or hysterical, and the seat of the neurosis is in the brain.

In postdiphtheritic paralysis some branches, especially the pharyngeal and laryngeal, of the pneumogastric nerve may be involved.

While, as already said, trauma does not often act as a cause of irritation or paralysis of the pneumogastric nerve, yet surgical operations about the neck may involve this nerve or one or other of its branches. This is true, especially of ligation of the internal carotid artery.

In epilepsy and hysteria the pneumogastric nerve is evidently temporarily involved in the fits; thus the epigastric aura of epilepsy and the "globus" of hysteria are both manifested in the territory of this nerve, although the primary excitation is probably in its centre in the brain cortex.

Very few evidently of the fibres of the pneumogastric nerve are under the control of the will, if we except those that innervate the pharynx and the larynx. Most of its influence apparently is upon the functions of organic life. These functions, however, are influenced to a considerable degree by the emotions, and some of the most important examples of this occur as morbid phenomena. It is difficult, however, to make an exact estimate of these. We may only surmise that many of the cardiac and gastric disorders due to various mental states act through the pneumogastric nerve. Many of the so-called instances of reflex irritation of the stomach and many cases of nausea and vomiting from injuries or diseases of remote organs evidently act by way of this nerve. Thus vomiting is not an uncommon symptom of organic diseases of the brain such as tumor and abscess. In such a case the symptom is probably caused by irritation of some of the association tracts or of the nucleus or trunk of the nerve itself. In

diseases of the kidneys and in pregnancy and diseases of the uterus, vomiting is not an uncommon symptom, and is probably caused by irritation either directly of the fibres of the pneumogastric nerve or indirectly by way of the sympathetic.

The symptoms of affection of the pneumogastric nerve are either irritative or paralytic. These will be considered in turn in reference to the various branches of the nerve trunk.

The *pharyngeal* branches of the pneumogastric nerve, as already explained, go to form, with branches of the sympathetic and of the glossopharyngeal nerves, the pharyngeal plexus. This plexus may be involved in various ways. One of its commonest affections is seen in postdiphtheritic paralysis. It is also implicated in the combined sclerosis known as labio-glosso-pharyngeal paralysis. Other affections of it are rare. Meningitis, tumors at the base of the brain, and disease of the bones might cause paralysis of the pharynx, but certainly this is not common. The symptoms of paralysis of the pharyngeal muscles are embarrassment or loss of power of swallowing. Liquids cause much difficulty, as they regurgitate through the nose, especially in cases of associated paralysis of the muscles of the soft palate. Particles of solid food also may pass into the larynx. A semi-solid food is best swallowed. In severe cases food may lodge and remain in the pharynx, and serious results may occur from strangling and choking. If minute particles of food pass into the lung a dangerous form of pneumonia may result. Spasm of the pharyngeal muscles is a rare symptom of involvement of the pharyngeal plexus. It is seen in hydrophobia, in which case it may possibly be due to irritation of the nerve trunks or nerve endings. It possibly occurs in the initial stage of the epileptic paroxysm and is a very common symptom in hysteria, although the true "globus" of hysteria is probably an affection rather of the œsophagus than of the pharynx. In the occupation neurosis of professional voice-users, a spasmodic action of both the pharyngeal and laryngeal muscles occurs. The pharynx, as Wyllie[9] has pointed out, plays an important part in the production of voice. In the disease known as clergyman's sore throat there is often a well-marked neurotic element in which the action of the associated muscles of the larynx and pharynx in the production of voice is impaired. In some of these neurotic cases the voice, in the midst of public speaking or singing, suddenly leaves the patient, as the result of a spasmodic action of the muscles of phonation. An hysterical element is not uncommon in some of these cases. The true aphonia of hysteria, however, is a psychic rather than a local affection.

The *laryngeal* branches of the pneumogastric nerve are probably more frequently involved in disease than any others. They are

especially liable to irritation and pressure from tumors and aneurysm in the neck and thorax, and to wounds inflicted during surgical operations. As already explained, the two laryngeal nerves—superior and recurrent or inferior—supply the larynx with sensory and motor filaments. The former of these filaments run largely in the superior, the latter in the recurrent branch. The superior laryngeal nerve, however, supplies motor fibres to the cricothyroid as well as to the lower constrictor muscle.

The opening and shutting of the glottis are affected by antagonistic muscles. These muscles, however, are supplied by fibres that run through the single nerve trunk—the recurrent laryngeal. These fibres, of course, are distinct and arise from distinct nerve cells, although passing through the same trunk. The fact that they do pass through the same nerve trunk, which may be variously affected by irritative and paralyzing lesions, is one of great clinical importance. The two main functions of the glottis are concerned first with respiration, second with phonation. During respiration the glottis is widened, or, more properly, it is widened during inspiration. It is closed firmly to prevent the entrance of foreign bodies into the larynx, and it is closed also immediately before the act of coughing. During phonation the laryngeal muscles give the vocal cords a certain tension, and at the same time the latter are approximated to each other. The tension of the vocal cords is produced by the cricothyroid muscles, which are supplied by the superior laryngeal nerves, but all the other muscles concerned with the functions of the glottis are supplied by the recurrent nerves.

Considering these facts, when a vocal cord stands at continued rest in the position normally occupied during phonation, that position may be due either to spasm or fixation of the muscles which close the glottis on the one hand, or to paralysis of the muscles which hold the glottis open on the other hand (Solis Cohen[11]). This phenomenon, according to Cohen, has been usually attributed to paralysis of the nerve supplying the posterior cricoarytenoid muscle, the function of which is to keep the vocal bands asunder, to hold the rima glottidis open for the needs of ordinary respiration, and even to draw these bands open to their widest extent for the needs of extraordinary inspiration. Cohen says that this view is supported by the fact that the muscle, in almost every case in which it is paralyzed by pressure upon its nerve, is found post mortem to be degenerated, and that little if any similar atrophy is found in the antagonistic muscles. This view is supported by Semon, who concludes that organic disease of the root or trunk of the motor nerve of the larynx (*i.e.*, the recurrent nerve) is earliest and sometimes exclusively manifested in paralysis

of this dilator muscle, *i.e.*, the posterior cricoarytenoid. Cohen himself, however, inclines to the view first expressed by Krause, of Berlin, who believes that organic irritation of the recurrent laryngeal nerve produces spasm of all the muscles (both the dilator and constrictor) of the pharynx—*i.e.*, of those that preside over the various acts of respiration, phonation, and coughing. Inasmuch, however, as the constrictor muscles preponderate over the dilator, the spasmodic closure of the glottis occurs in spite of the fact that the antagonistic or dilator muscles may also be thrown into spasm. This view of Krause seems to me to explain best some of the well-known facts of the clinic. In most lesions which cause irritation of the recurrent laryngeal nerve, such as tumors and aneurysms, we must suppose that both sets of fibres—*i.e.*, those passing to the dilator as well as those passing to the constrictor muscles—are irritated. This is due to the fact that the fibres for both sets of muscles are included in the one nerve trunk. As, however, in all such cases spasmodic closure and not spasmodic opening of the glottis is the rule, it seems that the only inference is that the muscles that preside over closure of the glottis overmaster those that preside over its opening, and that therefore spasmodic closure occurs, and we have the well-known paroxysmal dyspnœa which is so characteristic of pressure upon the recurrent laryngeal nerve. The danger in most cases in which irritation occurs to the laryngeal nerve is from spasm and closure of the glottis. Paralysis of this, especially when it occurs only on one side, is not as a rule sufficient to cause obstruction to the passage of air into the lung, for the reason that the unparalyzed side affords sufficient space. As the cricothyroid muscles are the chief muscles for rendering the vocal cords tense, and as these are supplied by the superior laryngeal nerve, this element in phonation is not always involved in affections of the larynx or its nerve supply. As all the other muscles, however, are supplied by the recurrent laryngeal nerve, paralysis or irritation of this nerve may variously affect the voice. According to Landois and Stirling, paralysis of the motor nerves of the larynx causes aphonia or loss of voice. If the tensors of the vocal cords are paralyzed, *i.e.*, the cricothyroid muscle, monotonia is the chief result. In paralysis of the larynx disturbance of respiration may not be important so long as respiration is tranquil, but so soon as increased respiration occurs, due to exercise or exertion, dyspnœa sets in because of the inability of the glottis to dilate. If only one vocal cord is paralyzed, the voice becomes falsetto. Sometimes the vocal cords are only paralyzed to such a slight degree that they do not move during phonation, but do move during forced respiration and during coughing. In many cases in which the function of the recurrent laryngeal nerve is

interfered with by aneurysm or tumor, the vocal cord is seen on the paralyzed side to be at rest in the position of phonation. To conclude, the important symptom caused by irritation of the motor nerve of the larynx is spasmodic closure of the glottis. This is a clinical fact about which there need be no speculation. Paralysis of these motor nerves, on the other hand, causes various affections of phonation, such as monotonia, falsetto tone, and various other defects of voice. It may also cause dyspnœa, which is only marked during exercise owing to the forced respiration thus rendered necessary. In other words, a paralysis which leaves the chink of the glottis only partially closed may give sufficient space for gentle respiration but not sufficient for the forced respiration required by exercise.

The dyspnœa caused by pressure upon the recurrent laryngeal nerve is not always merely paroxysmal. It may be, for reasons already explained, more or less constant, especially during exertion. Thus in a case reported by Solis Cohen, a man, aged forty-four years, with aneurysm of the arch of the aorta and of the left subclavian artery, had fixation, probably due to spastic immobility, of the left vocal band in the phonatory position. His voice was weak and diphthonic, the higher pitch tone being metallically shrill and squeaky. Respiration was embarrassed on exertion, but during rest in a sitting position it was quiet, at a rate of from 20 to 24 per minute. The laryngoscope revealed tense immobility of the left vocal band in the phonatory position. The aneurysm in this case compressed the inferior laryngeal nerve on the left side as it winds around the arch of the aorta. Cohen attributes the fixed position to a preponderating spasm of the group of adductor muscles rather than to paralysis of the abductor. As we have seen, however, some other observers consider this to be a true paralytic phenomenon. Cohen reports other cases in which this fixation of the vocal band in the phonatory position was due to other causes. Thus in one case it was caused by a large thyroid body, and in another by a left-sided pleuritic effusion with displacement of the heart. He thinks that the displacement of the heart had caused a strain upon the recurrent nerve by dragging the arch of the aorta forwards, downwards, and to the right. This author says that complete destruction of the conductivity of the recurrent laryngeal nerve leaves the vocal band immobile in a position which does not interfere with the respiratory function. In conclusion, it seems that the gravest dangers from interference with the function of the motor nerve of the larynx are two in number: first, spasm producing paroxysmal or even continued dyspnœa; and second, paralysis of the dilator muscles which, while permitting respiratio

during rest, causes dyspnœa during exercise, owing to the inability of the glottis to dilate.

The left recurrent laryngeal nerve is rather more liable to be involved by aneursym than its fellow, owing to the fact that it curves around the arch of the aorta.

The paroxysms of dyspnœa caused by involvement of the recurrent laryngeal nerves are occasionally fatal. Budd[a] reports the case of an old man who had paroxysms of difficult breathing. He had a small tumor on the left side of the neck close to the trachea. Occasionally he had violent attacks of suffocative dyspnœa, and in one of these he died. The autopsy revealed a small malignant growth situated in the cleft between the larynx and the trachea on the left side, and involving the recurrent laryngeal nerve. Neither the larynx nor the trachea had been subjected to mechanical pressure or organic obstruction.

Hale White[b] has recently advanced the theory that the recurrent laryngeal nerve has a trophic function for the thyroid gland. He reports two cases of aneurysm of the aorta with involvement of the recurrent laryngeal nerve, in which there was marked atrophy of the thyroid gland. The changes in the gland were degenerative and destructive, in one case the gland being little else than a mass of ill-formed fibrous tissue. White suggests that this fact is important in view of the possible causation of myxœdema in similar cases, but he records no observation to prove that myxœdema occurs under such circumstances.

Morell Mackenzie[a] recorded a case of paralysis and atrophy of the abductor of the left vocal cord, caused by laryngeal growths pressing upon the recurrent laryngeal nerve. There was extreme dyspnœa and stridulous breathing on the slightest exertion, but these ceased when the patient lay down and also when he went down-stairs. The autopsy revealed a number of small tumors growing outside of the windpipe midway between the larynx and the bifurcation of the trachea. In one of these the left recurrent nerve was completely embedded, and where it emerged it was seen to be red and inflamed. The left abductor or cricoarytenoid muscle was found to be pale and atrophied, and on microscopical examination its fibres were seen to have undergone complete fatty degeneration. No other muscle was unhealthy. This case seems to prove the correctness of the view that paralysis and degeneration of the abductor muscle is the cause, in some of these cases at least, of the dyspnœa. This is so especially in this type of case, in which the dyspnœa is increased on exertion and is not spasmodic and paroxysmal in time. The fact, however, that this muscle alone is involved when the whole nerve trunk, containing as it does

fibres to many other laryngeal muscles, is so seriously implicated, is difficult to understand and requires further explanation.

Newman[100] has laid down some general rules as to the diagnostic significance of laryngeal symptoms resulting from pressure by aneurysms upon the vagus and its recurrent laryngeal branch. These rules briefly are as follows: 1st. Aneurysm of the aorta and of the innominate artery may give rise to laryngeal symptoms only, so that it may require critical physical examination to form a positive diagnosis. 2d. In the early stage pressure may cause paroxysms of most urgent dyspnœa, accompanied by laryngeal stridor and paroxysmal cough. 3d. At a later stage paralysis, limited usually but not always to one side, occurs, characterized by phonative waste of breath and imperfect cough, but without dyspnœa except when reflex spasm is indicated on the opposite side. According to these rules it seems that spasm of the adductor muscles causing paroxysms of dyspnœa is more likely to be an early symptom, and is due to irritation of the recurrent nerve, but that later in the case paralysis of the abductor may occur, and this does not lead to dyspnœa, except, as already explained, during exercise or exertion, or when associated with spasm of the adductors on the opposite side. These rules possibly are subject to many exceptions, but in the main they seem to be based upon the true condition seen in these cases. They indicate, however, the necessity for extreme caution in arriving at a diagnosis of the true cause for either laryngeal spasm or paralysis.

The treatment for the various affections of the recurrent laryngeal nerve is not as a rule satisfactory. As the causes for the irritation or paralysis of the nerve trunk are usually tumors or aneurysms in the neck or thorax, but little can be done. Tumors at accessible points should of course be excised. The principal means for relief in urgent paroxysmal dyspnœa is tracheotomy. Anodyne and sedative medicines are but of little if any value in these cases. The surgical treatment for these various disorders of the recurrent laryngeal nerve cannot be discussed at length in this work.

The next important branches of the pneumogastric nerve are the cervical and thoracic *cardiac* branches. These join branches from the sympathetic to form the cardiac plexus. The pneumogastric nerve through these cardiac branches exercises an inhibitory action upon the heart; it also supplies the heart with sensory fibres. In some animals it is supposed that the heart receives also some of its accelerator fibres through the vagus. According to Landois and Stirling, feeble stimulation of the pneumogastric nerve sometimes causes an acceleration of the beats of the heart, but this probably occurs only when accelerator fibres are conveyed in its trunk. The depressor

nerve of the heart, but not properly its inhibitory nerve, is the nerve of Cyon, arising by one branch from the superior laryngeal, and usually by a second from the trunk of the vagus itself. The true inhibitory nerve arises from the inferior cardiac (Landois and Stirling). Stimulation of the inhibitory nerve causes a slowing of the heart's action and an increase in the force of the heart beat. When the central end of one vagus is stimulated, provided the other vagus is intact, the heart may be arrested reflexly in diastole. Stimulation of the cardiac branches of the vagus may cause attacks of suspension of the beats of the heart, accompanied by anxiety and a feeling of impending death. Goltz proved that attacks of this sort may be produced reflexly by tapping the intestines. On the other hand, paralysis of the cardiac branches of the pneumogastric nerve causes marked acceleration of the pulse to 140, 160, or even 200 beats per minute. In extreme cases the pulsation is very irregular in force and rhythm.

It is difficult to estimate the exact rôle of the pneumogastric nerve in diseases of the heart. As already said, this nerve is no doubt affected by some of the poisons, especially tobacco and alcohol. Nicotine and atropine are known to physiologists as paralyzants of the inhibitory fibres of the vagus nerve. In chronic poisoning by tobacco the disorder of the cardiac muscle known as the "tobacco heart" is probably, in part at least, an affection of the inhibitory fibres of the pneumogastric nerve. This affection is characterized by irregularity in the action of the heart, palpitation on exertion, some dyspnœa, and a sense of pain or constriction about the cardiac region of the chest. These symptoms are such as would be caused by the depressing action of a poison upon the vagus or its cardiac branches.

In alcoholic multiple neuritis a very common symptom, as already stated, is tachycardia or rapid action of the heart. In some of these cases the heart beats at a rate of 100, 120, or, as I knew it in one instance, 140, in spite of prolonged rest in bed. In these cases I have observed that the heart is also very rebellious to such cardiac stimulants as digitalis; in fact, I have failed a number of times to secure the characteristic action of digitalis under these circumstances. This condition is not without danger, and in some cases of alcoholic neuritis is the immediate cause of death. Thus I have known a patient suffering with alcoholic multiple neuritis in all four extremities and with wandering delirium and tachycardia, die suddenly from heart failure. In these cases we perhaps cannot estimate with scientific precision the exact condition of the pneumogastric nerve and its inhibitory branch. Accurate microscopical research is much needed in this direction. Still from the mere clinical standpoint it

seems justifiable to infer that these cardiac symptoms are due to involvement of the vagus or its cardiac branches. Instances are on record (Gowers) in which the vagus or its cardiac branch has been involved in enlarged glands or even in a small tumor. In these cases extreme frequency of the pulse has been observed.

Finally, the pneumogastric nerve contains afferent or sensory fibres, and these may be involved in some disease processes. It is possible that the pain of angina pectoris is caused, in part at least, by irritation of these sensory fibres. Pseudo-anginal attacks have been noted in cases, for instance, in which the cardiac plexus has been involved, as in a tumor.

The pulmonary branches of the pneumogastric nerve probably have some action in disease. They supply the muscular fibres of the bronchioles, and it is possibly through their mechanism that asthma is caused. Experiments have shown that congestion, pneumonia, or even gangrene of the lungs follows section of the vagus nerve. Section of both vagi, according to Landois and Stirling, is followed by diminution in the number of respirations. The pneumonia which occurs under these circumstances has been observed since the time of Valsalva. It may be explained by the fact that loss of motor power and sensibility of the larynx occurs as well as loss of sensibility of the trachea, bronchi, and lung. Hence the glottis is not closed during swallowing, and foreign bodies, such as small particles of food, enter the respiratory passages. In animals this pneumonia has been prevented by the insertion of a tube into the trachea through an opening in the neck. The occurrence of pneumonia is also favored by the fact that the labored and difficult respiration causes congestion and pulmonary œdema. Finally, these changes may be promoted by interference with trophic functions in the vagus. Rabbits as a rule die very quickly from pneumonia after section of the vagi. The presence of distinct trophic fibres in the pneumogastric nerve, however, is still somewhat problematical. This function in this nerve, as in all nerves, probably resides in the motor and sensory neurons.

The œsophageal branch of the pneumogastric nerve forms with its fellow of the opposite side the œsophageal plexus. We have no very distinct knowledge of the action of these branches in disease. They preside in health over the peristaltic action of the œsophagus and supply sensory fibres to the upper part only of this tube. Their action is entirely reflex or associative in character and is not properly under the control of the will. The sensation of "globus" in hysteria is possibly due to disorder of the œsophagus and pharynx. When the œsophagus is the seat of any irritative lesion, such as trauma, foreign

body, or neoplasm, its reflex action is probably manifested and may account in part for the obstructive symptom. It is impossible, however, as a rule in such cases to distinguish between the symptoms of mechanical obstruction and those due to reflex spasm. In some cases, however, of slight injury to the pharynx and œsophagus a well-marked neurotic element is present and should be recognized at its true worth. For instance, a slight injury such as may be caused by an irritating fluid or a foreign body, may be the starting-point for a most obstinate spasmodic action of the œsophagus on attempts at swallowing. Food is regurgitated, and the patient frequently suffers greatly in his general health. In such cases we may suppose that a slight persistent lesion exists and is the starting-point for an obstinate reflex neurosis. Such cases have been reported by Gull, Pepper, and others. They often merge into true cases of hysterical vomiting, in which this symptom persists probably long after the initial lesion has healed. Thus in a case reported by the writer[61] the vomiting had been caused by the swallowing of an irritating fluid. It had continued for many months and had reduced the patient to a condition of such extreme emaciation that her life was endangered. In these neurotic cases the vomiting is usually accomplished without effort and without nausea. It is in fact not so much an act of vomiting as of regurgitation. The food, for instance, does not always enter the stomach, but is rejected from the œsophagus. Hence this symptom is sometimes called *œsophagismus*. This subject has been discussed at length by Lasègue. In extreme cases the efforts at regurgitation continue even when there is no food in either the gullet or stomach, the patient merely bringing up a little tenacious mucus. These cases can usually be distinguished from cases of organic disease by a consideration of the cause of the malady, the character of the vomiting, the age of the patient, who is usually young (although not always), the absence of cachexia, and the presence of other hysterical stigmata. In these neurotic cases success can sometimes be obtained by suggestive therapeutics. Treatment with drugs is as a rule unsatisfactory.

The gastric branches of the pneumogastric nerve are probably not infrequently irritated by diseases of the stomach. The fibres are both sensory and motor. By the former probably all morbid sensations from the stomach are conveyed to the brain and vomiting is no doubt excited through them. The motor fibres preside over the movements of the walls of the stomach. The pain from organic disease of the stomach, and the various gastric neuralgias, are no doubt located in the pneumogastric nerve. Very rare cases have been reported in which disease at the root of the nerve has caused such

symptoms as excessive appetite, vomiting, etc. In some cases, however, hunger and thirst have been abolished by disease of the vagus (Johnson). The gastric crises of locomotor ataxia are probably due to irritation of the sensory neurons, whose cell bodies are located in the jugular and plexiform ganglia on the trunk of the nerve near its superficial origin. Pressure on the trunk of the nerve is said to have caused vomiting.

Although the pneumogastric nerve has connections in the abdomen, as, for instance, with the liver and intestines, distinct symptoms in these organs have not been noted in its diseases.

The *treatment* for diseases of the pneumogastric nerve or its more important branches must depend of course upon their causes. Some of this treatment has already been indicated. In organic disease, causing irritative or paralytic symptoms, such as tumors of the neck and anterior mediastinal space and aneurysm of the aorta and great blood-vessels, little can be done except by surgical means, and not always then, for relief. Drugs as a rule are of but little importance in any affection of the vagus. In case of syphilitic meningitis at the point of exit of the nerve from the medulla, antisyphilitic treatment should be given a full trial. Such cases are extremely rare, however, and their successful treatment is probably still rarer. As I have already said, the poisoning by alcohol of the cardiac branches of the pneumogastric nerve is exceedingly rebellious to treatment. Only prolonged rest, full diet, and small doses of strychnine give good results. Digitalis in my observation does not exert its full physiological effect in these cases. A recumbent position is imperative, as the risk of death from cardiac failure in alcoholic multiple neuritis is not to be ignored. In the œsophagismus or reflex neurosis of the œsophagus due to an initial irritative lesion of this tube local treatment, in my observation, is not useful and is sometimes strongly contraindicated. Exceptions occur, of course, in cases in which the initial lesion is persistent and accessible, but the majority of these cases need simply wise management and proper mental impression. The gastric branches of the pneumogastric nerve may be involved in so many lesions of the stomach that it is impossible here to outline a complete treatment for all these cases. This is best considered under the head of the respective lesions. In very severe gastralgia and in the crises of locomotor ataxia strong anodyne drugs alone give relief. The most important of these are morphine and other opiates, cocaine, croton chloral, atrophine, aconitine, and chloroform internally. Electricity in any of its forms is of little use in any of the affections of the pneumogastric nerve.

Diseases of the Eleventh Nerve.

The eleventh or spinal accessory nerve is exclusively motor. It arises from the lower part of the medulla oblongata and from the upper part of the spinal cord. Hence anatomists distinguish two

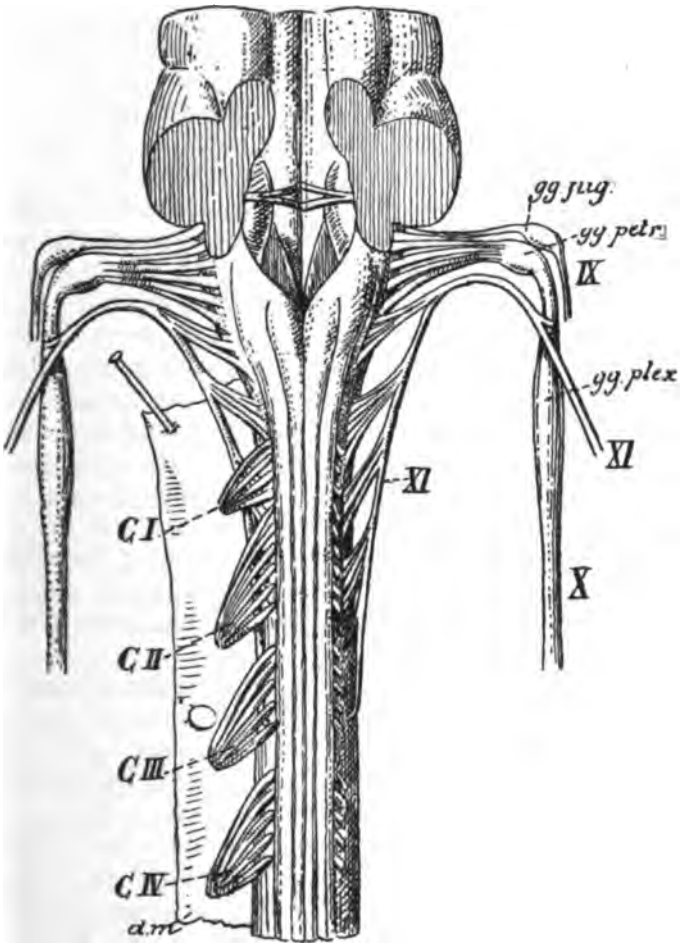

FIG. 27.—Apparent Origin of the Accessory Nerve of Willis, of the Pneumogastric, and of the Glossopharyngeal Nerve. (Van Gehuchten.)

portions of the nerve: the bulbar and the spinal. The nerve is a motor nerve similar in type to the other motor nerves of the spinal cord with the exception that it has more numerous and more widely located roots of origin. It is supplied exclusively to the sterno-

cleidomastoid and trapezius muscles. The spinal portion of this nerve arises from large ganglion cells in what is known as the lateral horn of the cervical cord from the level of the first to that of the third and sometimes fourth cervical nerves. The roots emerge from the spinal cord on its lateral aspect. The fibres of the bulbar portion of this nerve arise from cells of origin in what is called the nucleus ambiguus, which is in fact a column of gray matter which exists in the interior of the whole length of the medulla oblongata and is analogous to the anterior horn of the spinal cord. The fibres from the spinal portion of this nerve unite into one nerve trunk. This ascends in the spinal canal up to the level of the bulb, entering the cranium through the foramen magnum, and unites with the bulbar portion. The roots of origin of the bulbar portion arise from the same sulcus behind the olivary body from which arise the roots of the ninth and tenth nerves. This, the accessory or bulbar portion of the nerve, is the smaller of the two. The single nerve trunk, thus formed from the spinal and bulbar portions, passes from the cranium by the jugular foramen. As it emerges from the cranium the nerve divides into two main branches. The first of these, or internal branch, passes entirely to the pneumogastric nerve between its two ganglia, *i.e.*, the jugular and plexiform ganglia. This branch is described as formed by the fibres of the nerve which have come from the bulbar or accessory portion. The external branch, supposed to be of spinal origin, descends in the same sheath with the tenth nerve. It pierces the sternocleidomastoid muscle, supplying it with motor filaments, and then passes obliquely across the suboccipital triangle, entering beneath the anterior border of the trapezius, to which muscle it is distributed (Gray, Van Gehuchten).

The spinal accessory may be affected by disease or accident in various ways. Its nucleus or series of nuclei may be the seat of degeneration along with the nuclei of other motor nerves, as, for instance, in anterior poliomyelitis and amyotrophic lateral sclerosis. Its two main trunks of origin—the spinal and the bulbar or accessory—may be involved in meningitis or caries of the bone at the base of the brain. One or both of them may be damaged by a tumor in the superior cervical or bulbar region. Hemorrhages in these parts are not common. After its passage from the jugular foramen the nerve may be injured by tumors, abscesses, or wounds in the neck. Injury to the nerve where it passes across the space between the sternomastoid and the trapezius muscles may be inflicted by the pressure of heavy weights carried or falling upon the shoulders. Inflammation due to exposure to cold, such as is seen sometimes in the facial nerve, is not common in the spinal accessory nerve.

The *symptoms* of disease of the spinal accessory nerve may be due either to irritation or paralysis. The former causes spasm; the latter loss of power in the muscles supplied by the nerve. The distribution of the paralysis depends of course upon the point of the lesion. In case the nuclei are degenerated, as in amyotrophic lateral sclerosis, both the sternocleidomastoid and the trapezius muscles may be progressively paralyzed. In cases, however, of this disease and of progressive muscular atrophy the upper part of the trapezius muscle is sometimes the very last to lose power and volume and hence was called by Duchenne the *ultimum moriens*. The explanation of this fact is that the trapezius muscle is supplied only in this upper part to any extent by the spinal accessory nerve; the rest of its innervation coming from the spinal nerves. Hence in cases in which the upper part of the trapezius and the sternomastoid do not degenerate the inference is that their nuclei in the lateral column of the cervical cord do not degenerate so readily as the nuclei of the other spinal nerves that are affected. In cases in which the nerve trunk is involved anywhere in its course in the upper cervical spinal canal, in the cranium, or in the neck, the paralysis of course involves both the sternomastoid and the trapezius muscles. In these cases, however, the sternomastoid muscle is completely paralyzed, while the trapezius muscle is completely paralyzed only in its upper portion, although its other portion may be paretic. This, as already explained, is due to the fact that the trapezius is supplied in part by the spinal nerves.

Paralysis of the sternocleidomastoid muscle interferes with rotation of the chin towards the unparalyzed side. The paralysis, as a rule, is visible only on attempts at moving the head. At such times the inability to rotate the chin away from the paralyzed side and the flatness and inactivity of the paralyzed muscle are very apparent, whereas during perfect rest there may be no deformity visible except in long-standing cases. In these latter the paralysis, combined with wasting of the muscle, causes a loss of characteristic contour of the neck. Paralysis of the upper part of the trapezius, *i.e.*, the part which originates from the occipital bone, also causes alteration in the shape of the neck and loss of power, which are notable especially on deep inspiration. Although the main body of the trapezius muscle is not paralyzed, still it is rendered somewhat paretic by loss of function of the spinal accessory nerve. The shoulder falls slightly and the scapula rotates slightly inwards, due especially to the unopposed action of the rhomboid muscles. As under these circumstances the deltoid muscle loses some of its basis of support, elevation of the arm is effected awkwardly and with some difficulty.

In some instances of basilar meningitis both spinal accessory nerves are involved. This may be seen in tuberculous meningitis in children. In such cases the patient may be unable to hold the head upright, the paralysis of the sternomastoids permitting it to fall backwards, while that of the trapezius permits it to fall forwards. Hence the head is remarkably devoid of support, falling about in various directions when the patient is lifted upright. In some cases of amyotrophic lateral sclerosis paralysis of the trapezius and associated muscles permits the head to fall forwards with the chin resting upon the sternum in a characteristic way. The patient may not have lost absolute control of the nodding movement, but after the head has passed a certain point in bending forwards it suddenly loses its support and falls forwards on the chest. This movement may be likened to the snapping shut of the blade of a penknife. In cases in which the nerve trunk is injured after it has passed through the sternomastoid muscle, *i.e.*, in the suboccipital triangle, the trapezius muscle is of course the only one involved. In all cases of degenerative disease of the nuclei or trunk of the spinal accessory nerve or of injury to its trunk the reactions of degeneration sooner or later display themselves. Exceptions occur in such slowly degenerative diseases as progressive muscular atrophy. When the nerve trunk is injured by pressure or wounds the reactions of degeneration appear promptly. The motor points of the muscles are located at the seats of entrance of the nerve into the muscle, and as these are superficial and hence readily accessible the reactions of degeneration may be very readily studied.

It has been commonly assumed in the past that the accessory or bulbar part of this nerve supplies the laryngeal muscles through the branch which passes from the trunk of the spinal accessory to the pneumogastric. Hence it is claimed that when the spinal accessory nerve is involved in its intracranial portion there is also paralysis of the vocal cord. Recently, however, this origin of the motor fibres of the vocal cord has been denied and there is reason to believe that they proceed from the pneumogastric nerve proper. (See section on Pneumogastric Nerve.)

The *treatment* of paralytic affections of the spinal accessory nerve will depend upon their cause. In nuclear lesions little can be done to stay the progress of degeneration, which usually invades the nuclei also of many other nerves. These progressive degenerative diseases are usually obstinate to treatment. Strychnine, electricity, and a supporting diet offer the best prospects of staying the progress of the disease, but even this treatment is usually disappointing. In syphilitic meningitis at the base of the brain an antisyphilitic treat-

ment is of course indicated. In tuberculous meningitis practically no treatment avails. In tumors, abscesses, caries of the bones, and wounds of the neck the treatment is largely surgical, but is not always available. In strictly peripheral lesions, such as are caused by blows, pressure, and other injuries of the nerve trunk, cure is often obtained. In these cases electricity applied to the sternocleidomastoid and trapezius muscles is the best. This can be supplemented by hypodermic injections of strychnine into the substance of the muscle.

The following case illustrates very well paralysis and atrophy of the muscles supplied by the spinal accessory nerve in amyotrophic lateral sclerosis. The patient is under the writer's care in the Philadelphia Hospital.

FIG. 28.—Paralysis of the Trapezius Muscles in a Case of Amyotrophic Lateral Sclerosis. (Philadelphia Hospital.)

A. G——, Italian laborer, aged 50 years, with a history of syphilis of fourteen years' standing, began three and a half years ago to have weakness of the arms, coming on gradually. He now has marked atrophy of the muscles of the arms, of the forearms, shoulders, neck, and trunk. The atrophy is so great that many of these muscles have apparently completely disappeared. There is no fibrillation. The sternomastoid and trapezius muscles (as can be seen in the illustration) have almost entirely disappeared. Their paralysis and loss cause marked dropping of the head forwards upon the chest, as well as backwards. The sternomastoid, in fact, can only be made out as a thin fibrous cord. The patient has exaggerated knee-jerks and a feeble and spastic gait. Sensation is not involved and the pupils react normally. There is no muscular atrophy of the legs. In this case the affection of the spinal accessory nerve is undoubtedly nuclear and the nerve is involved with many other nerves, especially those arising from the cervical enlargement of the spinal cord. The diagnosis of amyotrophic lateral sclerosis is indicated in this case by the muscular atrophy of the upper limb and shoulder girdle, by the spastic and feeble gait, by the absence of sensory symptoms, and by the exemption of the bladder and rectum.

Torticollis.

Spasmodic affection of the muscles supplied by the spinal accessory nerve are seen especially in the disease known as torticollis or wry-neck. In this affection, however, it is not uncommon for other muscles beside the sternomastoid and trapezius to be affected; hence this disease cannot properly be said to be a disease exclusively of the spinal accessory nerve. As, however, it is shown most conspicuously in the two muscles supplied by this nerve it cannot be ignored in discussing the pathology of the eleventh nerve.

Spasm of the sternomastoid, trapezius, and associated muscles of the neck is an obscure affection so far as its essential causes and pathology are concerned. It has been attributed to cold, to overexercise, and to injury. The relative importance of these causes cannot be accurately estimated with our present knowledge. In some cases an initial stage of pain and stiffness in the affected muscles seems to indicate that the lesion, whatever it is, is irritative or inflammatory in type.

Wry-neck or torticollis usually begins gradually; in very rare cases, however, there may be a sudden explosion, as it were, of the disease. In the usual form, however, there may be an initial stage of pain and tenderness in the affected muscles, while these become stiff and the head consequently is held in an awkward, painful position. This spastic or tonic stage after a while gives way to the intermittent or paroxysmal stage, although the exact type varies in different cases. In some cases, for instance, there is always more or less spasticity and contracture of the affected muscle, and this is aggravated at short intervals by paroxysms in which the muscles contract even to their full extent. In other cases the type is more purely intermittent or paroxysmal. The contraction may almost if not quite relax, the patient's head assumes a normal position, then in a few moments the muscles begin again to contract, passing through a full stage of extreme physiological activity. In some cases this contraction is steady and tonic; in others it is clonic, the muscles jerking the head in a series of short, even rhythmical contractions.

The exact muscles affected in torticollis vary greatly. As a rule, however, one or other, or both muscles supplied by the spinal accessory nerve are involved. The disease, in fact, may be said to expend its greatest force through this nerve. In many cases, however, it is not confined to them, other muscles of the neck, face, shoulder, and even upper arm being implicated. Occasionally it is confined to only

one muscle; when this is the case the muscle involved is usually the sternocleidomastoid.

The *symptoms* depend upon the muscles involved. When the sternocleidomastoid is the chief muscle affected the head is drawn downwards and backwards and towards the affected side, while the chin is protruded forwards and upwards towards the other side. When the trapezius is involved the shoulder is somewhat elevated and the head drawn still further backwards. Double torticollis, in which the muscles on both sides are involved, is occasionally seen. In this form the head is drawn directly backwards towards the shoulders, the face being held upwards. This is called retrocollic spasm and is usually seen in children. It is associated with other symptoms of brain disorder and is probably due to meningitis or some irritative lesion at the base of the brain or in the cerebellum.

As a rule torticollis is not painful. In most cases, however, it is naturally associated with great mental depression, as the patient is not only annoyed by it but is even prevented from attending to many of the ordinary avocations of life.

In cases which begin early in life there is often an apparent deformity in the side of the head and neck. Wilks claimed that facial asymmetry may be seen on the contracted side. This side of the head seems to be smaller than the other and the eye to be lower (Hamilton).

The course of torticollis is usually obstinate and progressive. The prognosis, however, especially in young persons, is not necessarily bad. Recovery occasionally occurs under judicious treatment or even spontaneously.

The *pathology* of torticollis is as yet quite obscure. The disease is usually claimed to be due to some affection of the spinal accessory nerve, but as it frequently extends and involves muscles not supplied by this nerve this explanation is not tenable in all cases at least. Explanation has been sought in the possible irritation of the nerve roots or the intracranial course of the nerve trunk by meningitis, but observations, as a rule, are lacking in confirmation of this. It has even been suggested by some that the lesion in some cases may be cortical; in other words, that the motor centres for the affected muscle in the brain cortex may be involved, but this explanation is based largely upon speculation. In a very few cases autopsy has revealed some gross lesion, such as meningitis or a tumor upon or within the medulla. Such cases, however, are atypical. In the great majority of cases autopsy has revealed nothing. It is even possible that the lesion may be in the muscles themselves, acting as an irritant on the nerve endings.

The *treatment* of torticollis with drugs is often most unsatisfactory. Morphine, gelsemium, conium, cannabis indica, chloral, the bromides, and other sedatives, give, as a rule, only temporary relief. The danger from their use is that the patient may become addicted to them. This is true especially of morphine and chloral. Gelsemium is highly lauded, but its value has been distinctly overestimated. In early cases perfect rest in bed should be enjoined for a long time. This gives the muscles complete rest and is a useful adjuvant to drugs. Electricity is of no value whatever in this disease. Apparatus intended to hold the head forcibly at rest may seem to do temporary good, but, as a rule, such apparatus cannot be worn for a long period. It will not stop the spasm and becomes very irksome to the patient and irritating to the parts upon which it makes efficient pressure. Surgery offers relief in some of these cases. The tendons of the affected muscles have been divided in some cases with benefit. The most common operation is division of the spinal accessory nerve; in some cases the nerve has been merely stretched. According to Keen[105] the nerve may be reached by an incision either along the anterior or posterior border of the sternocleidomastoid muscle. Keen has also performed resection of the posterior cervical nerves for torticollis. In three of his four cases the result has been good. These nerves supply the splenius, the rectus capitis, and the obliquus inferior muscles. The operation is somewhat complicated and need not be described here in detail. From what has been said, it follows that division of the spinal accessory nerve alone is not sufficient to cure torticollis in all cases. This is because, as already explained, the disease is not always confined to the muscles supplied by this nerve.

Diseases of the Twelfth Nerve.

The twelfth or hypoglossal nerve is exclusively motor and is supplied to all the muscles of the tongue. Its deep or nuclear origin is in the gray substance of the medulla oblongata, just beneath the floor of the fourth ventricle close to the median line. The cells of origin of the nerve are of the motor type. Their axis cylinders pass transversely forward and through the medulla, passing through a portion of the olivary body, and leave the bulb by the fissure between the pyramid and the olive. The protoplasmic processes of these cell bodies (*i.e.*, the dendrons) interlace with each other, and the more internal ones interlace with those of the opposite side, constituting thus a commissure between the neuclei of the two nerves analogous to that which is found between the multipolar cells of the spinal nerves

(Van Gehuchten). The nerve fibres in these two nuclei form a dense plexus and are no doubt in part the terminal and lateral arborizations of fibres coming from other systems of neurons deeper within the nervous system to put these in contact with the peripheral motor neurons of the hypoglossal nerve. Many of these doubtless are the terminations of the axis cylinders coming by way of the pyramidal tract from the motor cells of the cerebral cortex of the opposite side. Other of these fibres probably form connections with sensitive tracts having relations with the pneumogastric, the glossopharyngeal, and the trigeminal nerves. According to Van Gehuchten it is not yet proved whether or no there is any decussation between the fibres of the hypoglossal nerves. Such decussation does not exist between the motor spinal nerves. Duval and Kölliker deny such a decussation for the hypoglossal nerve.

Froriep and Beck[111] claim to have found a well-marked dorsal root for the hypoglossal nerve in artiodactyles, carnivora, and some other groups. This root is much reduced in size in certain other groups, and is absent in some. The hypoglossal nerve thus resembles a spinal nerve. The first cervical nerve shows a poorly developed dorsal root. These authors, however, do not claim that a dorsal or sensory root to the hypoglossal nerve is found in man.

The superficial origin of the hypoglossal nerve is by ten or twelve small fasciculi which issue from the medulla oblongata, as already said, in the fissure between the pyramid and the olive. These unite to form two main roots, which penetrate separately the dura mater and then unite to form one nerve trunk, after their passage from the skull by the anterior condyloid foramen. The nerve descends in the neck to a level with the angle of the jaw in close proximity to the pneumogastric nerve. It then passes forwards close beneath the occipital artery and to the outer side of the common carotid. Its terminal muscular branches are distributed to the styloglossus, hypoglossus, and geniohyoid muscles.

The anastomosing branches of the hypoglossal nerve are as follows: First, with the superior cervical ganglion of the sympathetic; second, with the plexiform ganglion of the pneumogastric nerve; third, with a loop joining the first two cervical nerves. According to Holl, quoted by Van Gehuchten, this anastomosis carries fibres from the cervical nerves to the hypoglossal nerve, and these probably pass to the thyrohyoid and geniohyoid muscles. Fourth, there is an anastomosing branch with the lingual or gustatory branch of the fifth nerve. It is from these numerous anastomoses that the hypoglossal nerve, originally motor, receives sensory filaments.

The principal branches of the hypoglossal nerve are the descend-

ing branch (called by English anatomists the *descendens noni*), the recurring branch, the branch to the anterior belly of the omohyoid, a branch to the thyrohyoid, and a branch to the geniohyoid. Finally its terminal branches pass to all the muscles of the tongue.

From this distribution it is seen that the hypoglossal nerve is the motor nerve not only of the tongue but of some of the muscles, both elevator and depressor, of the hyoid bone. The fibres, however, which supply the depressors of the hyoid bone are, according to Holl, as already said, not derived from the hypoglossal nucleus but from the first two cervical nerves. The action of these various muscles is first to move the tongue in different directions, both protrusion and retraction, and also in its various movements in the actions of rolling the bolus in the mouth during chewing and swallowing; second, to depress and elevate the hyoid bone in the movements of deglutition.

The hypoglossal nerve may be affected by various diseases and accidents. Its nucleus in the medulla oblongata may be the seat of degenerative changes, but these are usually associated with similar changes in the nuclei of other nerves, especially the glossopharyngeal. This degeneration is seen especially in the affection known as labio-glosso-pharyngeal paralysis. The nucleus of the nerve may also be involved by hemorrhages or tumors in the medulla. The fibres of origin of the nerve may be involved in meningitis, especially syphilitic, and by tumors or caries of the bone at the base of the brain near the exit of the nerve. The trunk of the nerve after it passes through the cranium may be injured by wounds and by various surgical lesions of the neck, as tumors, abscesses, etc. The trunk of the nerve, however, is rarely, if ever, the seat of either a simple or specific neuritis. The tongue is not infrequently paralyzed by cranial lesions above the nucleus of the hypoglossal nerve in the medulla. For instance, one-half is not infrequently paralyzed in hemiplegia from hemorrhage or embolic softening involving the motor fibres in the internal capsule. But all such cases must be distinguished from those that arise from strictly peripheral lesions, which alone concern us here.

The *symptoms* of involvement of the nucleus or trunk of the hypoglossal nerve are paralysis of the side of the tongue to which the nerve is supplied. The paralysis is entirely motor, as the hypoglossal nerve supplies no sensory fibres to the tongue or neighboring parts. When one-half of the tongue is paralyzed the symptoms are highly characteristic and easily recognized. On protrusion the tongue deviates towards the paralyzed side. This is for the obvious reason that the unopposed muscles of the unparalyzed side push the tongue not only out of the mouth, but being unop-

posed propel it towards the side of the lesion. The movements of the tongue within the mouth are also conspicuously embarrassed. The movement of the paralyzed side is of course defective. Chewing is done awkwardly because the patient cannot keep the food between the teeth on the unparalyzed side. Still in some cases this defect is not so apparent as would be supposed. Swallowing may also be done awkwardly, for the reason that the tongue cannot propel the food with its normal strength and precision into the pharynx. In swallowing liquids, especially in the act of sucking, this deficient power of the tongue may be more apparent. In cases of nuclear degeneration of the hypoglossal nerve the lesion is almost without exception bilateral. In these cases the tongue lies inert and flabby on the floor of the mouth and all its normal activities in chewing, swallowing, and articulation are impaired. Speech under these circumstances is much interfered with, all the speech sounds depending on the proper use of the tongue being of course abolished or almost abolished. Finally in peripheral lesions of the hypoglossal nerve, *i.e.*, in lesions of its nucleus or trunk, trophic changes occur. The tongue wastes, becomes fissured or puckered into folds, and its electrical reactions are altered. Thus in severe cases faradic contractility may be entirely abolished, but the reactions of degeneration to galvanism, although probably present, are rather difficult to demonstrate. Taste is not truly abolished by paralysis of the hypoglossal nerve, but it is somewhat blunted by the inability of the tongue to roll sapid substances over its surface.

E. Ballard [104] relates a case of paralysis of the hypoglossal nerve followed by sloughing of the tongue. The patient, an old man aged seventy-eight, had a sudden paralysis of the right side of the tongue associated with speech defects and occipital pain. Extensive sloughing, which resulted in loss without hemorrhage of the entire right side of the tongue, occurred. The patient eventually recovered. No mention is made of hemiplegia, but the case is rather imperfectly reported. The presence of aphasic symptoms would indicate a central lesion, but, on the other hand, the rapid and extensive sloughing of the tongue would seem to indicate an involvement of the hypoglossal nerve or its nucleus.

Habershon [105] reports a case of cancer of the cervical vertebræ with paralysis of the right hypoglossal nerve. The patient protruded the tongue towards the paralyzed side, and that side of the tongue was flaccid and wrinkled. She had difficulty in managing her food. She was obliged to keep it in the middle of the mouth; if she let it pass to the side her tongue could not deal with it. The sternohyoid muscle on the right side was also paralyzed. This was especially notable

when the muscle was exercised in raising the head; the left sternohyoid then started forwards, but the right was not seen and a deep hollow was left between the sternomastoid muscle and the larynx. This muscle also failed to contract when the patient swallowed. Electro-contractility was abolished on the right, but preserved on the left side of the tongue. There was also slight paralysis of the facial nerve. The autopsy revealed a cancerous mass involving the basilar process, and compressing the nerves mentioned.

The *diagnosis* of paralysis of the hypoglossal nerve is so clearly indicated by the paralysis of the one side of the tongue that it can scarcely be mistaken. The diagnosis of the exact lesion that causes the paralysis and of its precise location may, however, be a matter of some difficulty. In paralysis of one-half of the tongue, due to a central lesion (*i.e.*, a lesion above the nucleus of the hypoglossal nerve), there are always associated symptoms which point more or less clearly to the site of the lesion. Thus in hemorrhage or embolic softening in one cerebral hemisphere—as, for instance, that which occurs so frequently in the internal capsule—the hemiplegia and facial paralysis are even more conspicuous symptoms than the paralysis of the tongue. In all central lesions, moreover, the paralysis of the tongue is not associated with muscular atrophy and reactions of degeneration in that organ. Moreover, the character of the paralysis itself admits usually of some distinction. In peripheral palsies, *i.e.*, those due to lesions of the nucleus or nerve trunk, the paralysis is more nearly absolute than in cases of central lesions. Nuclear disease of the hypoglossal nerve is usually bilateral, but a unilateral lesion involving the hypoglossal nucleus might be associated with a hemiplegia of the opposite side, thus presenting a crossed hemiplegia and hypoglossal paralysis. This would be so, however, only in case the lesion was located above the decussation of the pyramids. In case of a bulbar lesion, all the fibres of the nerve might be involved, consequently all the muscles, not only of the tongue, but of the elevators of the hyoid bone, would be paralyzed. In cases of lesions in the neck, in which the nerve is injured below any of its main branches, the muscles supplied by these branches would of course escape. Thus if the descendens noni were not involved, the omohyoid, sternohyoid, and sternothyroid muscles would escape. These muscles also would escape in case the nerve were injured by any lesion above its anastomosis with the superior cervical nerve, because the fibres of this descendens noni are probably supplied entirely from the superior cervical nerves. In degenerative lesions of the nuclei of the hypoglossal nerve the affection is bilateral and associated with similar lesions in the

nuclei of some of the other bulbar nerves. The cardinal distinction, finally, of a nuclear or peripheral lesion of the hypoglossal nerve is the muscular atrophy associated with the reactions of degeneration.

The *prognosis* in any case of paralysis of the hypoglossal nerve depends, of course, upon the nature of the lesion. As a general rule the prognosis of the peripheral palsy (of which we here alone speak) from whatever cause is unfavorable. Nuclear degeneration of the hypoglossal nerve as it occurs in labio-glosso-laryngeal paralysis is an inveterate and progressive malady, and as a rule is but little influenced by any treatment. In gross lesion at the base of the brain, such as tumor, hemorrhage, bone disease, and meningitis, the prognosis is also necessarily unfavorable. Even in syphilitic meningitis treatment is often disappointing.

In cases of surgical lesions of the neck involving the hypoglossal nerve, the *treatment* of course will be surgical, according to the nature of the lesion. If it is thought worth while to apply electricity, this can be done by means of an appropriate electrode introduced into the mouth and pressed directly upon the tongue. Strychnine is possibly a useful drug in some cases of nuclear degeneration, but it too is often disappointing.

DISEASES OF THE SPINAL NERVES.

Morphological Note.

The spinal nerves present a special type, in this respect differing from most of the cranial nerves. This type consists in their having anterior or ventral and posterior or dorsal roots. The anterior root is composed almost entirely of motor fibres, which are, in fact, the axis cylinders of a series of peripheral motor neurons, whose cell bodies are located in the anterior horn of the gray matter of the spinal cord. The posterior root presents on its trunk a large ganglion, and is composed almost exclusively of sensory fibres, which are the axis cylinders of a group of peripheral sensory neurons which are located in this ganglion. Thus the anterior and posterior roots differ from each other both in their functions and in the point of origin of their fibres, the motor cells being located within the spinal cord, while the sensory cells are located outside of the spinal cord in the ganglion of the posterior root. Although, as just said, the anterior root is essentially motor, yet it must be recalled that a few sensory fibres are included in it, which are derived from the ganglion on the posterior root and which reach the anterior root by way of the junction of the two. The anterior and posterior

roots unite a short distance after leaving the spinal cord, to form one common nerve trunk. This junction is peripherad to the ganglion on the posterior root, which is situated just within the intervertebral foramen. The only exception to the general plan or type just described is in the posterior root of the first cervical nerve, which frequently has no ganglion upon it (Gray). As noted above, Froriep and Beck have found a posterior or dorsal root of the hypoglossal nerve in certain animals, and there is also a tendency of the first cervical nerve to develop this root imperfectly.

The motor neurons whose fibres form the anterior root have their cell bodies in the anterior horn of the gray matter of the spinal cord. The axis cylinder, leaving its cell body, pushes forwards through the white matter and appears upon the anterolateral surface of the cord, whence it is continued through the nerve trunk to its point of distribution in the muscle. Its dendritic or protoplasmic processes, freely anastomosing with those of its fellows in the spinal cord, constitute the receptive fibres for the set of deeper neurons whose cell bodies are located in the brain cortex. Some of these dendrons pass to the opposite side of the cord through the anterior white commissures, thus establishing connections but not true anastomoses with the motor cells of the two sides (Van Gehuchten, Kölliker, Ramon y Cajal).

The sensory neurons whose axis cylinders constitute the posterior root have an entirely different origin from the motor. Their cell bodies are located in the ganglion on the posterior root. From its cell body in this ganglion the neuron projects its axis cylinder forwards in the posterior root into the spinal cord. Towards the periphery it projects another process, which is also morphologically like a medullated axis cylinder, and this constitutes a sensory fibre in the spinal nerve and passes to its point of distribution in the skin or mucous membrane. When the axis cylinder of the sensory neuron passes into the posterior column of the cord, it divides into two branches, one of which ascends and the other descends. These fibres are variously distributed in the central nervous system. Some pass into the posterior gray horn. The longer tracts pass upwards through the column of Burdach, and then into the column of Goll, and thus to the nuclei of the posterior columns of the medulla (Jakob). These nuclei are the starting-point for a new series of sensory neurons, whose axis cylinders are projected upwards into the brain mass. The fibre passing from the cell body of the sensory neuron in the ganglion of the posterior root towards the periphery constitutes, as already said, the sensory fibre of the nerve trunk. It is medullated and in every respect resembles a motor axis cylinder. Some histologists have claimed, however, that this fibre represents

the dendron of the sensory neuron. It may be recalled here that Lenhossek demonstrated that the sensory neuron in the earthworm is located just within the epithelium of the skin, and that consequently its axis cylinder is properly the fibre which extends from this cell body towards the central nervous masses. By analogy it would seem that the true axis cylinder of the peripheral sensory neuron in the higher animals is the prolongation from the posterior ganglion into the spinal cord, and not the fibre from this ganglion through the nerve trunk to the periphery. While the posterior roots are described as essentially sensory, it must be recalled that they contain some fibres from cell bodies located in the posterior horn of the spinal cord, and that these fibres should be considered, according to Van Gehuchten, as centrifugal or motor fibres.

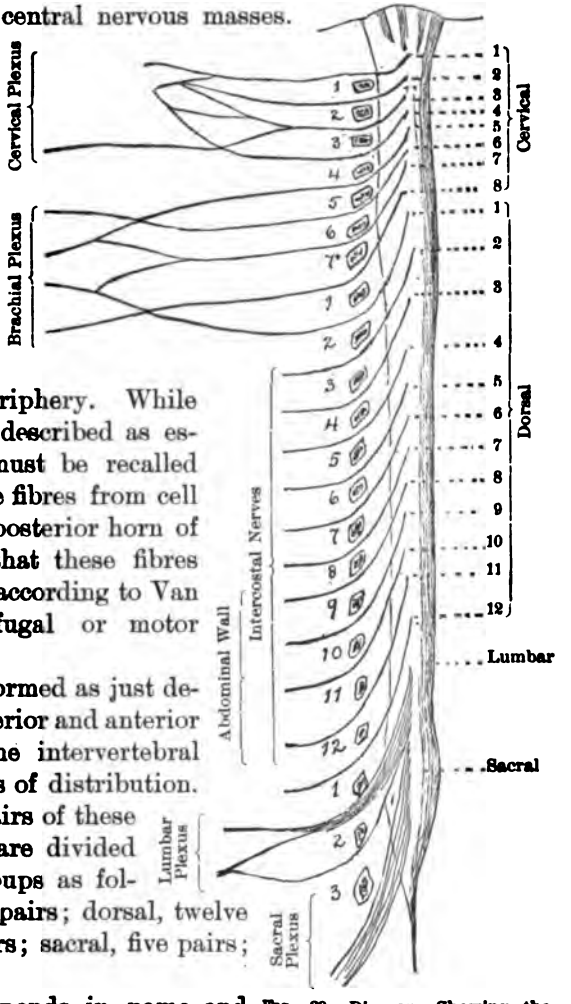

FIG. 29.—Diagram Showing the Groupings and Plexuses of the Spinal Nerves. (After Baker.)

The spinal nerves, formed as just described from their posterior and anterior roots, pass through the intervertebral foramina to their places of distribution. There are thirty-one pairs of these spinal nerves, which are divided by anatomists into groups as follows: Cervical, eight pairs; dorsal, twelve pairs; lumbar, five pairs; sacral, five pairs; coccygeal,* one pair.

Each group corresponds in name and number with the vertebræ, with two exceptions. The cervical group, for instance, comprises eight pairs. This is due to the fact that the first pair of spinal nerves is included in this group, although it leaves the spinal canal between the occipital bone and the arch of the atlas, thus giving

*There are in reality three pairs of coccygeal nerves, but two of them are rudimentary and are only microscopic in size.

to the group one more pair of nerves than there are cervical vertebræ. Again, in the coccygeal region there is but one pair of nerves, and not a pair for every vertebra in the coccyx.

The trunks of the spinal nerves do not all escape from the spinal canal exactly at a level with their points of origin in the spinal cord. With the exception of the first pair of cervical nerves, they all have a short descending intraspinal course, which increases in length for each pair of nerves from above downwards. Thus the first pair of cervical nerves passes out from the spinal canal between the occipital bone and the arch of the atlas after a very short almost transverse course across the spinal cavity, but the second pair bends slightly downwards before reaching the foramina between the atlas and the axis. The third pair has a still longer course within the spinal canal before reaching their foramina, and so on down the whole extent of the cord, the intraspinal course of each pair being slightly longer than that of the preceding pair. In the lower dorsal and especially in the lumbar region this intraspinal course of the nerve trunks becomes quite prolonged. The nerves of the lumbar enlargement of the cord, in fact, have such a long intraspinal course before reaching their respective foramina that they quite fill up the lower portion of the spinal canal far below the termination of the cord, and this intraspinal group of lumbar and sacral nerves is called the *cauda equina*.

The cervical, lumbar, and sacral nerves do not pass directly to their points of distribution, but in each group the members unite with each other to form a plexus, and this plexus gives origin to the various nerve trunks. These plexuses will be described in their proper places. The dorsal nerves, however, do not form a plexus, but each nerve trunk passes directly out to its point of distribution.

The course of the spinal nerves within the spinal canal is shown in the accompanying diagram (Fig. 29). This intraspinal course of these nerves must be thoroughly understood by the student of nervous disease, in order that he may be accurate in localizing lesions, both in the spinal cord and in the nerve trunks. It will be seen that practically none of the spinal nerves, excepting the first, arises in the spinal cord on a level with its point of exit from the canal. Thus, for instance, the fifth cervical nerves originating in the fifth spinal segment arise from the cord at a point on a level with the space between the third and fourth vertebræ, but leave the spinal canal by the foramina between the fourth and fifth vertebræ, and that this point of exit from the canal is still some distance above the spinous process of the fifth vertebra. Farther down the cord this intraspinal course is much prolonged. In the dorsal region the spinal nerves do not emerge from the canal until they have gone the distance

of almost or quite the length of two vertebræ. In the lumbar cord this intraspinal course is still greater. The cord itself terminates at a point midway between the first and second lumbar spines, but the lumbar and sacral nerves pursue a long course from this point through the lower end of the spinal canal until they reach their respective foramina. From this arrangement it is seen that some diseases within the spinal canal, especially at its lower end, are practically peripheral diseases, because they involve the trunks of the nerves and produce symptoms exactly similar to those that are produced by lesions outside of the spinal canal.

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD.

Segment.	Muscles.	Reflex and Centres.	Sensation.
First cervical.	Rectus lateralis. Rectus capitis. Anticus and posticus. Sternohyoid. Sternothyroid.		
Second and third cervical.	Sternomastoid. Trapezius. Scaleni and neck. Omohyoid. Diaphragm.	Hypochondrium (?). Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex and neck. (Occipitalis major, occipitalis minor, auricularis magnus, superficialis colli, and supraclavicular.)
Fourth cervical.	Diaphragm. Deltoid. Biceps. Coracobrachialis. Supinator longus. Rhomboid. Supra- and infraspinatus.	Pupillary (fourth cervical to second dorsal). Dilatation of the pupil produced by irritation of neck.	Neck. Shoulder, anterior surface. Outer arm. (Supraclavicular, circumflex, external-musculocutaneous, cutaneous.)
Fifth cervical.	Deltoid. Biceps. Coracobrachialis. Brachialis anticus. Supinator longus. Supinator brevis. Deep muscles of shoulder blade. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	Scapular (fifth cervical to first dorsal). Irritation of skin over the scapular produces contraction of scapular muscles. Supinator longus. Tapping the tendon of the supinator longus produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm to the wrist. (Supraclavicular, circumflex, external cutaneous, internal cutaneous, posterior spinal branches.)
Sixth cervical.	Deltoid. Biceps. Brachialis anticus. Subscapular. Pectoralis (clavicular part) Serratus magnus. Triceps. Pronators. Rhomboid. Latissimus dorsi.	Triceps (fifth to sixth cervical). Tapping elbow tendon produces extension of forearm. Posterior wrist (sixth to eighth cervical). Tapping tendon causes extension of hand.	Outer side and front of forearm. Back of hand, radial distribution. (Chiefly cutaneous, internal cutaneous, radial.)
Seventh cervical.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Serratus magnus. Latissimus dorsi. Teres major.	Anterior wrist (seventh to eighth cervical). Tapping anterior tendons causes flexion of wrist. Palmar (seventh cervical to first dorsal). Stroking palm causes closure of fingers.	Radial distribution in the hand. Median distribution in the palm, thumb, index, and one-half middle finger. (External cutaneous, internal cutaneous, radial, median, posterior spinal branches.)

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD.—*Continued.*

Segment.	Muscles.	Reflex and Centres.	Sensation.
Eighth cervical.	Triceps (long head). Flexors of wrist and fingers. Intrinsic hand muscles	.....	Ulnar area of hand, back and palm; inner border of forearm. (Internal cutaneous, ulnar.)
First dorsal.	Extensors of thumb. Intrinsic hand muscles Thenar and hypothenar muscles.	.....	Chiefly inner side of forearm and arm to near the axilla. (Chiefly internal cutaneous and nerve of Wrisberg or lesser internal cutaneous.)
Second dorsal.	.....	.....	Inner side of arm near and in axilla. (Intercostohumeral.)
Second to twelfth dorsal.	Muscles of back. Erectores spinæ.	Epigastric (fourth to seventh dorsal). Tickling mammary region causes retraction of the epigastrium. Abdominal (seventh to eleventh dorsal). Stroking side of abdomen causes retraction of belly. Vasomotor centres. Second dorsal to second lumbar.	Skin of chest and abdomen, in bands running around and downwards, corresponding to spinal nerves. Upper gluteal region. (Intercostals and dorsal posterior nerves.)
First lumbar.	None.	Cremasteric (first to third lumbar). Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum. (Iliohypogastric, ilioinguinal.)
Second lumbar.	Vastus internus.	Patellar. Striking patella tendon causes extension of leg.	Outer side and upper front of thigh. Lumbar region. (Genitocrural, external cutaneous.)
Third lumbar.	Sartorius; adductors of thigh. Flexors of thigh.	.....	Front and outer side of thigh. Inner side of leg and foot.
Fourth lumbar.	Extensors of knee. Adductors of thigh.	Gluteal (fourth to fifth lumbar). Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh, leg and foot. (Internal cutaneous, long saphenous, obturator.)
Fifth lumbar.	Outward rotators. Flexors of knee. Flexors of ankle. Peronei. Extensors of toes.	Achilles tendon. Over-extension causes rapid flexion of ankles, called ankle clonus.	Back of thigh and outer side of leg and ankle; sole; dorsum of foot. (External popliteal, external saphenous, musculocutaneous, plantar.)
First and second sacral.	Calf muscles. Glutei. Peronei. Extensors of ankle. Small muscles of foot.	Plantar (fifth lumbar to second sacral). Tickling sole of foot causes flexion of toes and retraction of leg.	Back of buttock and thigh, side of leg and ankle; sole; dorsum of foot.
Third, fourth, and fifth sacral.	Peronei. Muscles of bladder, rectum, and external genitals.	Genital centre. Vesical centre. Anal centre.	Circumanal region, anus, rectum, penis, urethra, vagina, perineum. (Small sciatic, pudic, inferior hemorrhoidal, inferior pudendal.)

The distribution of the spinal nerves can be best shown in tabular form. The above table, taken from Dana and founded upon observations of Starr, Mills, Thorburn, and others, gives the relations merely of each pair of cervical nerves by segments. The table does not show, however, the various nerve trunks which supply

individual muscles or muscle groups, since these individual nerve trunks arise (except in the dorsal region), not from the roots of the spinal nerves directly, but from the plexuses which are formed by the junction of the various groups of nerves. The table, however, is of value to the student of peripheral nervous diseases for the reason that it gives the relations of the various muscles, reflexes, and areas of sensation to the various spinal nerve roots. Hence it indicates what particular groups of sensory and motor neurons preside over the various muscles, reflexes, and sensory areas.

In describing the diseases of the spinal nerves I shall take them as nearly as possible in order from above downwards. The plexuses formed by the upper cervical, by the lower cervical and two first dorsal, by the lumbar, and by the sacral nerves will be described in turn, and the diseases which affect each one of these plexuses as a whole will also be described. As, however, many of the most important nerves of the body arise from these plexuses, they will be described in due order rather with reference to the plexus from which they arise than with reference to the individual spinal nerves through which their fibres can be traced. Thus the musculospiral nerve will be described as one of the branches of the brachial plexus, and the sciatic nerve as one of the branches of the sacral plexus. No special attempt will be made to indicate from what particular segment of the cord these nerves originate, and consequently by what particular spinal roots they leave the cord. It is well known that most of the large nerve trunks of the arms and legs contain fibres from several segments of the cord, and that consequently they cannot be described as originating from any particular spinal nerve. Moreover, the symptoms of diseases and injuries of the nerve trunks are shown entirely in their peripheral distribution. Consequently their affections, to be thoroughly understood, do not require as a matter of first importance an accurate knowledge of the location of the cell bodies of their neurons in the spinal cord and intervertebral ganglia. In cases, however, in which for any reason it may be necessary or desirable to trace the exact connection of any particular nerve trunk with any particular spinal root or spinal segment, this can be done by reference to the above table.

Diseases of the Cervical Plexus.

The cervical plexus is formed from the anterior branches of the four upper cervical nerves. The roots of these four cervical nerves increase gradually in size from the first to the fourth, and the posterior roots are much larger than the anterior in the proportion of three to one, except in the case of the first cervical nerve. This proportion is

much greater than at other levels of the cord (Gray). Each of these cervical nerves divides as it passes from the spinal canal into two branches, an anterior and posterior, and it is from the anterior that the cervical plexus is formed. The chief branches of the cervical plexus go to the skin about the neck and occiput. It supplies a few muscles with motor nerves, and these muscles are among those that are sometimes affected in torticollis. The chief branch of the cervical plexus is the phrenic nerve, which is supplied to the diaphragm.

Diseases of the cervical plexus as a whole are very rare. These nerve roots and the plexus which they form may be involved in a number of lesions. Caries of the vertebræ or at the base of the skull, abscesses, tumors, and other surgical lesions such as wounds, might occasionally involve some of these nerve trunks. They are involved also sometimes in torticollis, for, as already explained, this disease is not always confined to the muscles supplied by the spinal accessory nerve. In some general diseases, such as progressive muscular atrophy and amyotrophic lateral sclerosis, the muscles supplied by all the cervical nerves may be involved. Occasionally a tonic or spastic contraction of the muscles about the base of the skull is seen. This may be caused especially by meningitis. The nerve trunks forming the cervical plexus may also be involved in the grave forms of post-cervical neuralgia presently to be described.

Diseases of the Phrenic Nerve.

This nerve, also called the internal respiratory nerve of Bell, arises from the third and fourth cervical nerves. In descending through the neck it passes between the subclavian artery and subclavian vein. In the chest its most important relations are with the root of the lung, the pericardium, and the pleura. Upon reaching the diaphragm it divides and its branches separately pierce that muscle, to be distributed to its under surface (Gray). In addition to its terminal branches in the diaphragm the phrenic nerve gives small branches to the pericardium and pleura, and has connection with the sympathetic, and with the fifth and sixth cervical nerves.

The phrenic nerve, owing to its deep course, is well protected from external injury. Still it is occasionally impaired by blows and wounds of the neck, and sometimes in surgical operations on these parts. It may also be involved in caries of the bones of the spine. It is rarely if ever the seat of inflammation due to cold, but it may be involved in multiple neuritis, especially in those forms due to diphtheria and beriberi. In alcoholic multiple neuritis the phrenic nerve is possibly also sometimes involved.

Paralysis of both phrenic nerves causes complete paralysis of the diaphragm. This symptom, although quite characteristic, may readily be overlooked. As the diaphragm when paralyzed fails to descend with each inspiration, the abdominal contents of course do not descend; the upper part of the abdomen consequently does not protrude, but is inactive or even permanently retracted. A compensatory action of the other respiratory muscles is sometimes observed, causing increased activity of the movements of the upper part of the thorax, hence producing what is called the upper costal type of respiration. Bilateral paralysis of the phrenic nerve does not necessarily embarrass respiration while the patient is recumbent or inactive, but on exertion some dyspnœa results and even feebleness of voice. It is supposed that this paralysis also induces congestion of the base of the lung. Paralysis of one phrenic nerve causes little if any subjective symptoms, and may be overlooked because of the action of its fellow. But close observation will usually detect impaired movement of the diaphragm on the affected side. In multiple neuritis from alcohol, diphtheria, or beriberi, paralysis of the phrenic nerves is a serious complication. It is usually associated with some loss of power in the other nerves of respiration—those, for instance, supplying the thoracic muscles. Under these circumstances the thorax expands but little if any during inspiration, and the abdomen does not protrude. Danger to life is great from congestion and œdema of the lungs.

The *diagnosis* of paralysis of the phrenic nerve must be made especially from the failure of the descent of the diaphragm. This paralysis must be distinguished especially from hysterical rapid respiration and from diaphragmatic pleurisy. In hysteria rapid respiration, which is rather more apt to occur in women than in men, produces the upper costal type of respiration. In these cases there are usually other hysterical stigmata, and there is nothing in the case to indicate a cause for an organic lesion of the phrenic nerves. Moreover, there is no true dyspnœa in these cases, and the symptoms are apt rather to be relieved voluntarily by exertion than to be aggravated by it. That no true paralysis of the diaphragm exists in these cases can also usually be determined by securing the patients' attention and getting them to draw a long breath. Diaphragmatic pleurisy is associated with acute pain, and close observation will usually determine that the immobility of the diaphragm is caused by the inhibitory action of this pain. Moreover, the history of the case and the presence of some physical signs are usually sufficient to determine the diagnosis. Some authors have described a degeneration of the diaphragm itself, but this condition is certainly extremely rare and could not well be distinguished from paralysis of the phrenic nerve.

In fact, it is probable that degeneration of the diaphragm if it ever occurs is secondary to degeneration of the phrenic nerve itself.

No direct or specific *treatment* is available for paralysis of the phrenic nerve. When this paralysis occurs as a result of general poisoning of the peripheral nervous system, as, for instance, by alcohol, diphtheria, or the poison of beriberi, the treatment for the general condition is alone possible. This will be described in the proper place. Surgical lesions require surgical treatment, but this is often impracticable because of the gravity of these lesions in the deep course of the nerve.

The phrenic nerve can be stimulated with electricity by pressing one electrode deeply down behind the posterior margin of the lower end of the sternocleidomastoid muscle and placing the other electrode at any point along the edge of the ribs. Treatment with electricity, however, is of little value in paralysis of the diaphragm from any cause whatever.

Diseases of the Cervical Nerves.

A grave form of neuralgia, sometimes called postcervical neuralgia, is located in some of the branches of the upper cervical nerves. The chief of these branches to be involved is the occipitalis major. This is largely a sensory nerve, supplying the skin of the scalp as far upwards and forwards as the vertex. It is a branch of the second cervical nerve, and arises from the posterior division of that nerve and consequently not from the cervical plexus proper. It is a very large nerve and a much more important one from the pathological standpoint than it is usually considered.

Neuralgia of this greater occipital nerve and of its kindred branches from the upper cervical nerves and plexus is in some instances a very grave affection. The pain is felt especially in the distribution of the great occipital nerve, *i.e.*, over the occipital and posterior parietal regions. As in all grave neuralgic states, however, there are usually some points of extreme sensitiveness, and these exist especially where the nerve trunk and its main branches emerge from the deeper regions. The principal of these spots are: First, where the great occipital nerve pierces the trapezius muscle near its attachments to the skull; second, in the space between the trapezius and the sternomastoid muscle, where deep pressure affects the cervical nerves; third, over the boss of the parietal bone, where the nerve trunks are very superficial and easily affected by pressure.

The character of the pain in postcervical neuralgia is almost as severe in some instances as that which affects the fifth nerve. As a

rule, however, it is paroxysmal. It is a rare affection as compared with either trigeminal neuralgia or sciatica. It is sometimes associated with contraction of the muscles of the nape of the neck, but this contraction is probably voluntary, the result of the patient's effort to render the parts immobile and thus to secure as complete rest as possible.

The *causes* of postcervical neuralgia are frequently difficult to determine. The affection has sometimes been traced to a sprain or a bruise, as, for instance, from lifting or carrying heavy weights, or from a blow. In one very marked example which I once saw the paroxysm always appeared at the menstrual epoch and was evidently part of the nerve storm caused by dysmenorrhœa. In this case the pain was so severe as to cause cries of distress in the patient and to interfere entirely with sleep during at least one night. This form of neuralgia is probably associated in some cases with a gouty or rheumatic diathesis.

The *diagnosis* of postcervical neuralgia is usually not difficult. Mistakes, however, may occur. The most probable error would be to mistake caries of one or more of the upper cervical vertebræ for simple postcervical neuralgia. In caries, however, there is usually stiffness of the neck caused by rigidity of the muscles. Pain is felt on movement, on deep pressure, and on jarring the spinal column by causing the patient to rise on tiptoe and fall suddenly upon the heels. Moreover, the pain of spinal caries is not usually so remitting and paroxysmal as postcervical neuralgia. When once established it is more continuous and of longer duration. Finally, close observation will usually detect beginning deformity.

The *treatment* for postcervical neuralgia is very similar to that required in other forms of neuralgia. Anodynes will, of course, be demanded by the patient, but they should be given cautiously, lest some drug habit be formed. The alterative and antirheumatic remedies such as the iodides and salicylates should be given a trial. The coal-tar sedatives combined with the salicylates are sometimes efficacious. Thus in one case I knew great relief to be secured from small doses of antipyrin and salicylate of sodium. Morphine given under the skin will relieve the severe pain of the worst paroxysms. In some cases it may be worth while to try active counter-irritation; a fly blister is the best. The actual cautery, such as the Paquelin, is recommended by some, but is certainly a heroic remedy for this disease, especially its paroxysmal and remittent types. In grave, obstinately continuous forms, however, in which no other remedies avail, the Paquelin cautery might be used. This remedy, as is well known, sometimes gives most satisfactory results in other grave neuralgias,

as, for instances, sciatica. There can, therefore, be no objection to its use in severe postoccipital neuralgia, provided nothing else has given relief.

Diseases of the Brachial Plexus.

The brachial plexus is composed from the anterior branches of the last four cervical nerves and of the first dorsal nerve. It begins close under the lateral processes of the cervical vertebræ and extends to just beneath the clavicle, where it gives origin to its chief nerve

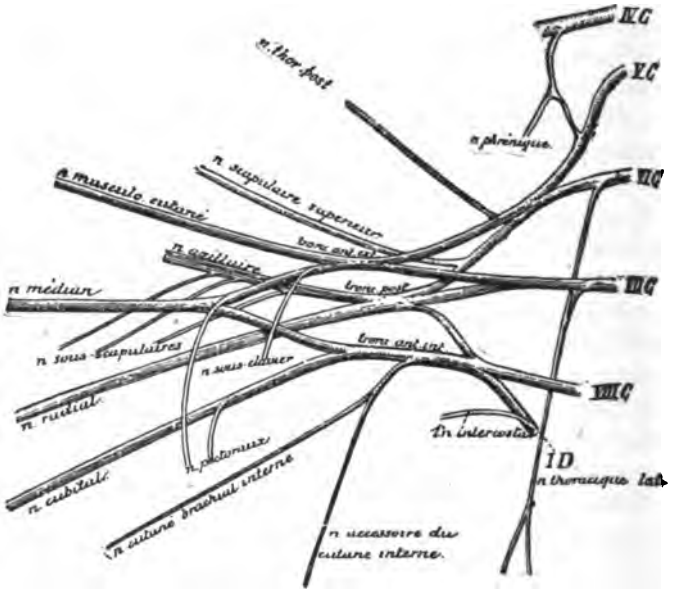

FIG. 30.—Diagram of the Brachial Plexus. (Van Gehuchten.)

trunks. It is triangular in shape, its base being situated at the vertebræ. The trunks of the five spinal nerves which go to form this plexus anastomose in such a way as to form three main trunks, two anterior and one posterior. The anterior trunks are named respectively the anterior external and the anterior internal trunk. The posterior trunk is called by that name. These trunks are formed as follows: The fifth and sixth cervical nerves unite to form one trunk which soon divides into an anterior and a posterior branch. The seventh cervical nerve before uniting with its neighbors divides also into an anterior and a posterior branch. The eighth cervical and first dorsal nerve unite and then immediately divide also into an anterior and a posterior branch. The three posterior branches unite with each other to form the posterior trunk, which subdivides into two terminal

nerves, the circumflex and the musculospiral. The anterior branch of the seventh cervical nerve unites with the anterior branch formed from the junction of the fifth and sixth cervical nerves to form the anterior external trunk, which subdivides into the musculocutaneous nerve and into another branch which forms with a similar branch from the anterior internal trunk the median nerve. The anterior branch of the eighth cervical nerve and of the first dorsal nerve forms the anterior internal trunk, which gives origin in turn to one of the branches constituting the median nerve, to the ulnar nerve, to the internal cutaneous nerve, and to the accessory branch of the cutaneous brachial internal nerve.

From this arrangement it is seen that the main nerve trunks of the arm and shoulder do not originate directly from the cervical and first dorsal nerves, but from the union of these nerves which forms the brachial plexus. Consequently each nerve trunk contains fibres from more than one cervical nerve. In consequence of this anatomical arrangement it is desirable to describe the diseases and injuries of the brachial plexus separately from the diseases and injuries of its individual branches, which form the main nerve trunks of the arm and shoulder.

The brachial plexus is sometimes the seat of severe neuralgia. In the majority of cases this brachial neuralgia is due to trauma or to some gross organic lesion, such as neoplasm. In all these cases the pathological state is probably one of inflammation of the nerve trunks. In some cases rheumatism and gout are probably exciting causes. The affection is usually unilateral and is not associated as a rule with paralysis of motor fibres. To this rule, however, there are exceptions. For instance, in traumatic cases the motor fibres as well as the sensory may be involved. As in most cases of severe neuralgia, painful points are felt in regions where the nerve trunks are most exposed or most easily subjected to pressure. These points are found especially on the side of the vertebræ, in the axilla, on the scapula, and on the ulnar and radial nerves in the forearm. There may be areas of paræsthesia and anæsthesia in the distribution of the various nerves, also some paralysis and muscular atrophy in cases in which motor fibres are involved. Trophic and vasomotor changes are also occasionally seen. These are practically the same that have already been described under the heading of trophic lesions of nerves. They are causalgia or burning pain, œdema, and glossy skin, and possibly herpetic and pemphigoid eruptions. When these latter occur they are evidences of inflammatory or irritative processes in the nerve trunks, and a diagnosis of brachial neuritis rather than simple brachial neuralgia is indicated. From the symptoms, in fact,

reported by various observers it seems that these cases of involvement of the brachial plexus must vary widely in their underlying pathological changes. In some cases, due possibly to rheumatism, gout, or other dyscrasia, the sensory phenomena predominate, and the condition may be called more properly a true neuralgia, in which gross permanent changes in the nerve trunks are lacking. In other cases, however, especially those of traumatic origin, in which there are associated trophic changes and paralyses, contractures, and muscular atrophy, the nerve trunks are probably the seats of active inflammation. The same may be said of some few cases which may be due to the poison of lead or of syphilis. Finally, the brachial plexus, or rather the spinal nerve roots which contribute to its formation, is occasionally the seat of the fulminating pains of locomotor ataxia. In these cases the specific changes probably begin in the cell bodies of the sensory neurons, located in the ganglia on the posterior nerve roots.

Cases of brachial neuralgia, so called, which are caused by traumata are among the most obstinate in their course and most severe in their symptoms. They are usually in fact the result of gross injury to the nerve trunks and consequently are associated with all the symptoms of organic changes in nerves. Thus, in addition to the severe pain, there is usually paralysis, muscular atrophy, reactions of degeneration, contractures, and trophic changes; in other words, there is grave neuritis.

The distribution of the symptoms caused by severe organic lesions of the brachial plexus is as follows: There is brachial monoplegia. The arm is inert and falls heavily against the trunk. All voluntary movements, such as flexion, extension, abduction, adduction, rotation, pronation, and supination in the arm, forearm, and hand, are totally abolished. The shoulder, as has been pointed out by Huet and by Raymond,[106] may be elevated by the action of the trapezius muscle. The reason for this action of the trapezius is not altogether clear. Huet gives the following explanation: The arm, hanging inert along the thorax, tends, by its weight pressing upon the trunk, to cause a certain feeling of uneasiness. To obviate this feeling the patient, perhaps instinctively and unconsciously, elevates the shoulder by contracting the trapezius in such a manner as to carry the paralyzed arm slightly away from the side of the trunk. The trapezius remains thus strongly contracted. This observer thinks that this action is not that simply of an antagonistic muscle, in which, as we know, contracture is not uncommon when its antagonist is paralyzed. It seems in me, however, that the theory of antagonism is not inappropriate in this case. The monoplegia of brachial lesions is not

only total, as just explained, but the paralysis is flaccid and accompanied with abolition of the tendon reflexes. The sensory symptoms are in many cases first those of acute pain. Sometimes the painful sensations are indefinable. They may be paroxysmal and worse at night than in the day-time. They frequently interfere with sleep. They do not always necessarily follow the trajectory of any particular nerve, but are diffused. They may be excited especially by pressure on the muscles.

The anæsthesia caused by a total lesion of the brachial plexus is as follows: The anæsthesia is total for the hand and forearm, but follows a line drawn from the inner aspect of the arm just above the elbow obliquely upwards to the outer aspect of the arm at about the insertion of the deltoid muscle. This distribution is characteristic and follows strictly of course the projection of the sensory fillets of the brachial plexus. The tip of the shoulder, as is well known, is not supplied by this plexus but by the cervical plexus, and the inner aspect of the upper arm by the first three pairs of dorsal nerves.

If these cases, as usually happens, are caused by injuries which leave open sloughing sores the inference is that the nerve trunks, having been originally torn and stretched, become eventually the seats of aseptic neuritis. Such a case has been reported by Sands and Seguin.[106a] In this case a young man received a severe wound of the arm from the premature discharge of a cannon which he was loading. He had a badly lacerated wound of the thumb and hand with an extensive burn of the forearm and with fracture of the radius and ulna. The extensive burn interfered with the proper dressing of the fracture, and a false joint resulted. Anæsthesia in the hand was absolute, but its limits were not indicated. Symptoms of irritation of the nerves did not appear until the end of the third week, when pain showed itself in the range of distribution of the ulnar nerve. This pain rapidly became excruciating and henceforth constituted the predominant symptom in the case. The healing of the burn was apparently a slow matter, and the amputation of the thumb joint (which had become necessary) left a small ulcer. Anæsthesia extended as high as the upper part of the arm, and complete paralysis of the arm was gradually established. All modes of sensation were abolished, *i.e.*, tactile sense, thermal sense, and pain sense. In this case amputation of the arm was resorted to because of the extreme suffering of the patient and the uselessness of the member. After amputation electrical irritability of the nerve trunks, the median, ulnar, and musculospiral, was found entirely abolished, consequently the inference was that these nerves were degenerated. The amputation brought but temporary relief and eventually the brachial plexus itself was excised

by Sands. At the operation it was found that the nerves constituting the brachial plexus were matted together and their dissection was by no means easy. The fifth, sixth, and seventh cervical nerves were cut in a mass, a piece fully a quarter of an inch being excised. The double cord formed by the union of the eighth cervical and first dorsal nerves was also cut. Even in the fresh state at the time of the operation the excised portions were not normal in appearance. The surface of the section was yellow, showing hardly a trace of the fasciculi, and the neurilemma was thickened and injected. This neuritis, extending thus to the very cervical roots themselves, was apparently of septic origin, and was of such a character as was more likely to happen in the days before antiseptic surgery. The character of the wounds, one of which was lacerated, the other of which was a deep burn, was just such as to produce a lesion of this kind. The microscopic appearances of the excised nerve cords forming the brachial plexus showed them to be the seats of extensive interstitial neuritis. The authors believed that the primary injury to the nerves was one of complete disruption of the nerve trunks in the brachial plexus. As, however, the evidence of this was not found at the time of the operation, it seems more probable that the primary lesion was one merely of injury to the nerve fibres, and that the nerve trunks eventually became the seat of infection from septic wounds. The case is important as illustrating the causation, symptoms, progress, and results of surgical treatment in these cases. The patient was not cured. At the last note he still suffered from much burning and lancinating pain. This he referred to the amputated finger and hand. He also had the phenomena of "phantom" hand. The reason for failure in this case was evidently due to the fact that the inflammation had extended even above the roots of the cervical nerves at their exit from the intervertebral foramina, and that consequently the nerve cells of the sensory neurons in the posterior ganglia were probably the seats of active irritation. Such a case consequently would probably become as intractable as the ordinary case of tic douloureux.

A case of painful neuromata, of rare interest, involving the nerves of the arm and the brachial plexus has been put on record by Maury and Duhring.[106b] The patient had suffered paroxysms of intense pain from neuromata growing in the skin and evidently involving small branches of the nerves. These paroxysms occurred five or six times in a day and continued from ten to fifteen minutes. Life had become so unendurable that exsection of the brachial plexus was advised and submitted to by the patient. The operation was performed by Maury,

who exsected the outer and the inner trunks of the plexus, but for some reason the posterior trunk was not divided. As a result of this operation the patient was greatly relieved but not entirely cured. Pain continued to be felt, especially on top of the shoulder, which the authors attribute to the fact that the skin of this region was supplied by the third cervical nerve. The patient of course had paralysis and wasting of the muscles with reactions of degeneration and anæsthesia extending well up to the shoulder. The excised portions of the nerveas howed slight beginning interstitial neuritis. No reason is given by the authors for not cutting the posterior trunk of the brachial plexus from which springs the musculospiral and circumflex nerves, nor is it clearly indicated that the muscles and skin supplied by these nerves were exempt.

The statement by the authors that the arm was completely paralyzed is difficult to reconcile with the fact that this posterior trunk, which is one of the most important divisions of the brachial plexus, was apparently not divided. The microscopic appearances of the excised nerves in this case of Maury and Duhring indicated that the active inflammatory changes had not advanced fully as far as the brachial plexus. As the exciting lesions were mostly peripheral and widespread, being neuromata in the skin, it is probable that the active changes were in the neighborhood of these tumors. The operation, of course, was indicated at the only available place where all the nerve trunks come together, *i.e.*, in the brachial plexus. The fact, however, that cure was not complete was probably due, as already indicated, to the failure to divide all the nerve trunks. The pain persisting about the shoulder was no doubt due to the fact that the posterior trunk of the plexus was not divided, and that consequently the circumflex nerve which supplies the integument of the shoulder was not cut off. This seems to be the true explanation rather than the one suggested by the authors.

Raymond [100] has narrated a case of brachial monoplegia caused by compression from a hemorrhage beneath the pectoral and other muscles. This rare case happened in a man who had had malaria and had been subject to hemorrhages of various kinds since his youth. He drank to excess, and it was while lying in a drunken sleep, in which he compressed the arm, that the hemorrhage occurred. Raymond's theory was that a rupture of a blood-vessel occurred during his sleep. The symptoms were complete brachial monoplegia with severe pain and œdema. The retroclavicular portion of the brachial plexus was alone involved, as the nerves arising from the plexus above this point (for instance, those to the pectoral muscles, to the rhomboid, and to the scapula muscles) were not paralyzed.

Injuries to the brachial plexus are sometimes caused in the newborn child by the manœuvres necessary for the disengagement of the trunk in difficult labors. Bailly and Onimus,[108c] have reported a case of extensive damage to the brachial plexus in an infant from this cause. The head was properly delivered, but the trunk remained firmly fixed in the pelvis. It was finally disengaged by making traction on the axilla, but this was done at the cost of extensive injury to the brachial plexus and its main branches. After birth it was found that the arm was paralyzed, the only spontaneous movements being a feeble extension of the forearm, flexion of the fingers, and extension of the ring finger and little finger. In consequence of this the writers believed that the nerves most affected were the circumflex and the musculocutaneous, although from their description it would seem that all the nerves of the arm were more or less injured. In this case a careful study by Onimus demonstrated that the faradic contractility was diminished for the triceps, for the flexor of the hand, and for the extensors of the finger, except of the ring finger. The deltoid, the infraspinatus, the biceps, and the brachialis anticus gave no response. Improvement in this case was steady, and at the time of the report the authors expressed the belief that the paralysis would eventually disappear. They did not share the opinion of Duchenne, who gave a grave prognosis for this paralysis. Abandoned to itself he claimed that it became incurable. He seemed to think that restoration of nerves is accomplished in man the less promptly the younger is the patient. Onimus concludes that in the early months of life prospect for improvement in severe injury to nerves is not bad.

The *treatment* for brachial neuralgia is similar to that for neuralgia in other parts. In the very grave forms, such as have been related above, due to infectious neuritis or neoplasm, anodyne and sedative medicines in large doses will be required. Morphine will undoubtedly be the drug that these patients will come to crave and to rely upon. It is almost impossible in fact to prevent patients, suffering as much as Duhring's patient suffered, from becoming habitues of opium in some form. Fortunately these cases are rare, otherwise we should probably have clearer indications for treatment. Perfect rest is indicated and should be secured by fixation dressing of some kind. In the case reported by Sands and Seguin the patient dreaded movement to such an extent that he became wildly excited at the thought of it. Hot fomentations are sometimes grateful and even curative in the milder forms of brachial neuralgia. There is a type of this disease, sometimes seen, in which the pain is located more especially in one nerve trunk, and in which there is probably a gouty

or rheumatic taint. In such cases preparations of salicylic acid should be given a full trial, and these may be combined with phenacetin, antipyrin, antifebrin, antikamnia, or exalgin. Cases of this kind usually make a satisfactory recovery, although the duration of the disease may last for several weeks. In the milder cases or in the terminal stage of more severe cases a mild galvanic current applied along the course of the nerve trunk is sometimes beneficial. Counter-irritation, especially by blisters over the painful point, sometimes gives relief. Iodide of potassium is of little service in any form of neuralgia or neuritis, unless this be due to some syphilitic lesion. The same may be said of the mercurials. Bromide of potassium and chloral hydrate are hypnotic rather than anodyne in their action, and consequently are of little use in these cases. If used at all, they must be given in very large doses and their depressing and other unfavorable action is most undesirable. I have known good effects from croton chloral in some cases of severe neuralgia, and it might be tried in the form here considered. Aconitine is recommended by some.

The surgical treatment of the severe forms of brachial neuritis caused by trauma or neoplasms has already been referred to in the description of the cases quoted above. Such a procedure is of course always a last resort, and can be recommended only when all other treatment has failed and when the patient's life has become intolerable from his sufferings. Excision of the brachial plexus is a most heroic remedy. It maims the patient even worse than amputation of the arm, because it leaves him in possession of an arm that is worse than useless. There can be no doubt, however, of the propriety of the operation in such rare cases as those in which it was performed by Sands and by Maury. It is highly important, however, that the operation, if performed, should be radical and complete. All three main trunks of the brachial plexus should be divided, and a sufficient portion of each trunk removed to insure against its reunion.

In cases of injury of the brachial plexus in the child at birth, rest, warmth, and the use of mild currents of electricity are indicated. The prognosis in these cases is usually favorable, but a guarded prognosis should be given at the beginning.

Diseases of the Circumflex Nerve.

The circumflex nerve arises from the posterior trunk of the brachial plexus, in common with the musculospiral nerve. It contains both sensory and motor fibres. The former are distributed to the skin over the deltoid muscle, especially its lower and posterior portion.

The motor fibres supply the deltoid muscle and the teres minor. The nerve before its distribution divides into two branches, an upper and a lower. The upper branch, which is the more important from the pathological standpoint, winds around the neck of the humerus just beneath the deltoid muscle and supplies this muscle with its motor branches, which pass into its substance from the under surface. This branch also gives off sensory filaments, which supply the skin over the lower portion of the deltoid muscle. The lower branch gives motor fibres to the teres minor and also to the posterior part of the deltoid muscle. It supplies sensory filaments to the skin over the lower and posterior surface of the deltoid muscle. The action of the muscles supplied by the circumflex nerve is to raise the arm directly upwards from the shoulder and hold it in that position. Of the two muscles the deltoid is the chief actor. The teres minor rotates the head of the humerus outwards, and when the arm is raised it assists in holding it in that position and rotating it outwards (Gray).

The circumflex nerve is especially liable to injury from dislocations of the head of the humerus and from fractures of the neck of that bone. It is seldom if ever the seat of a simple neuritis, such, for instance, as is due to exposure to cold. The nerve or its nuclei is frequently involved in degenerative diseases which attack large muscular groups, such as progressive muscular atrophy and amyotrophic lateral sclerosis. It is possibly involved occasionally in rheumatic and other inflammations of the shoulder joint, and this accounts for the fact that sometimes in such inflammation there is paralysis and atrophy of the deltoid muscle. From its protected position it is rarely if ever injured by wounds, but it may be involved in severe contusions of and blows upon the shoulder. By far the most important cause, however, of paralysis of the circumflex nerve is dislocation of the shoulder-joint. So important is this, in fact, that the practitioner should never lose sight of the possibility, and being called to such a case should always explain to the patient and his friends the risk that he runs from such a complication. Unfortunately the evidences of this injury to the nerve are not always very apparent for several weeks after the accident. The weakness or paralysis of the muscle is disguised first by the pain and the surgical dressings. Later, however, when attempts are made to move the shoulder, it gradually becomes apparent that the deltoid muscle is paralyzed. At first the impairment of motion may be thought to be due to some stiffness of the joint, but if careful inspection is made it will be found that the muscle does not contract and that it is beginning to lose volume. A very early indication of this injury is abolition of the faradic con-

tractility in the muscle and the onset of the reactions of degeneration. These tests with electricity are of the utmost importance in cases of dislocation of the shoulder-joint, and should never be omitted. They permit of the establishment of a positive diagnosis at an earlier stage in the history of the case than any other symptom. This is due to the fact, as already stated, that inhibition of movement in the joint during the first few weeks may be thought to be due to pain and stiffness caused by injuries to the structures of the joint, and may in fact be partly due to these, and thus disguise the paralysis of the deltoid muscle. The importance of an early diagnosis in such a case needs no comment. Paralysis and atrophy of this muscle, following dislocation of the shoulder-joint, is a serious complication, and one that cannot be recognized too soon for the welfare of the patient and the reputation of the surgeon.

The symptoms of injury to the circumflex nerve are paralysis of the deltoid muscle and some paresis of the teres minor muscle, and occasionally, but not always, anæsthesia of the skin overlying the lower and posterior portions of the former muscle. It frequently happens, however, that there is no anæsthesia, and this is in accord with the well-known fact that injuries to mixed nerves frequently impair the motor much more than the sensory fibres. When the deltoid muscle is paralyzed, the movements of the arm are much restricted. The upward and outward movement of the arm at the shoulder is entirely abolished. Rotation of the head of the humerus outwards is also impaired by the paralysis of the teres minor. This restriction of the upward and outward movement of the arm impairs many other associated movements at the shoulder-joint, in which the deltoid muscle takes an active part. The arm hangs helplessly against the trunk, except for the movements of abduction, flexion, and extension, which are imparted to it by other muscles. In course of time atrophy of the muscle supervenes. In cases in which the injury to the nerve has been severe this atrophy advances to an extreme limit. The deltoid muscle becomes flattened, and even presents the appearance of being scooped out. This is so characteristic as to be unmistakable when once seen. This hollowed space extends from underneath the clavicle in front, around underneath the acromium process and the spine of the scapula. It seems as though in some cases the muscle has almost entirely disappeared. With the beginning atrophy—in fact, before this atrophy is apparent—the faradic contractility of the muscle is abolished. This may occur very early in the case, even as early as the first week. It usually precedes the onset of the reactions of degeneration. These reactions may come on in all their classical stages. There may

be at first increased galvanic contractility, followed by decrease with modal and serial changes. As already stated, these electrical changes are of great significance, both for diagnosis and prognosis in these cases. With the atrophy of the muscle there is likely to be some stiffness due to ankylosis of the joint. This is probably not a true trophic lesion, but is due to the injury to the soft structures of the joint at the time of dislocation. It is no doubt favored, however, by the nerve injury. This, for instance, causes such impairment of the movements of the joint that the slight nutritive changes set up by the injury progress more rapidly and with more serious consequences than they would in case the muscle were not paralyzed. In time adhesions may form, and these still further impair the power of movement at the joint. They are manifest on passive movements. The head of the humerus is then seen to be firmly attached to the scapula. As the arm is moved the scapula moves with it, as is shown especially by the excursions of its inferior angle. These movements of the scapula with the humerus are in marked contrast with its immobility in health. The ankylosis may be disguised by the paralysis and atrophy of the deltoid muscle, but the simple test of observing the associated movements of the scapula with the humerus reveals the true state of the joint. In case no fibrous adhesions exist, passive movements of the joint in all directions without movement of the scapula are readily obtained.

In the case of a lady who injured the circumflex nerve by dislocating the head of the humerus, the course of events as observed by me was as follows: While dressing in the morning the patient tripped and fell, extending her arm towards the wall for support. The head of the bone was driven with some violence from its socket downwards and forwards. The dislocation was reduced while the patient was under ether, and without difficulty in a short time. The joint remained painful for some days. When attempts at restoration of movement were made, however, as the pain subsided, it was found that the deltoid muscle was unable to contract. Complete abolition of faradic contractility was noted in a short time, and this was followed by the appearance of the reactions of degeneration. Rapid atrophy of the muscle set in and eventually became extreme, so that the space occupied normally by the deltoid was hollow. Fibrous adhesions soon formed in the joint. The progress of this case was slow and tedious. Adhesions in the joint required to be eventually broken up, and the substance and functions of the deltoid muscle were not restored for fully a year. The patient eventually made a complete recovery. This case illustrated perfectly the risks which a patient runs in dislocation of the shoulder-joint due to injury to the circum-

flex nerve, and also the fact that the prognosis of such cases need not necessarily be hopeless.

Raymond [107] has reported a curious case of paralysis of both deltoid muscles, caused by elongation of the two circumflex nerves. This stretching of the nerves occurred while the patient was sleeping upon his back, holding his arms elevated with his hands joined behind his neck. On rising in the morning the patient was unable to raise his right arm, and at the same time he had pain at the level of the deltoid muscle. The left arm was not paralyzed at first, but became so after a few hours. Among the initial symptoms were violent muscular shocks or contractions in the affected muscle. The symptoms were such as have already been described as due to injury of the circumflex nerve. There were partial reactions of degeneration and involvement of the sensory branch of the circumflex nerve which supplies the skin over the deltoid muscle. The unique feature of the case was the involvement of both circumflex nerves by stretching, due to the peculiar attitude of the patient. From a study on the cadaver Raymond concluded that the nerves had been injured by elongation by the forced attitude of the patient, whose circumflex nerves were probably unusually short, *i.e.*, did not pursue as winding a course as is usual.

The *treatment* of paralysis of the circumflex nerve should be by electricity to the deltoid muscle, by hypodermic injections of strychnine into the substance of the muscle, by inunctions, and by massage. Electricity is probably the most important of these agents. As faradic contractility is usually abolished, this current should not be used. Mild, alternating galvanic currents should be applied at least three times a week, and these with passive motion and massage may require to be persevered with for many weeks or even months. The fibrous adhesions should be broken up. In the case above mentioned a full year elapsed before the deltoid muscle was restored, but the fact that it was eventually fully restored proves that time and treatment will often accomplish much in these cases.

Diseases of the Musculospiral Nerve.

The musculospiral nerve arises from the posterior trunk of the brachial plexus, in common with the circumflex nerve. It is the largest branch of the brachial plexus (Gray). In its course along the arm it winds around the humerus in a special groove known as the spiral groove, just beneath the triceps muscle. Its course is from within, behind, to the outer side of the arm. When it reaches the external condyle it divides into two branches, the radial and the posterior in-

terosseous nerves. Before dividing into these terminal branches its main trunk supplies muscular branches to the triceps, anconeus, extensor carpi radialis longior, supinator longus, and brachialis anticus. Its cutaneous branches, three in number, supply the skin with sensation on the posterior aspect of the arm, the anterior aspect of the lower part of the arm, and the back part of the forearm. The actions of the muscles supplied by the main trunk of the musculospiral nerve in its course through the upper arm are to extend the arm, to supinate the hand, and to extend the hand at the wrist. As it supplies the brachialis anticus muscle also, it has some control over flexion of the forearm.

The musculospiral nerve may be injured by blows, wounds, and pressure. Although in the main part of its course it is situated deeply beneath muscular masses, its close relation to the bone makes it especially susceptible to injury from pressure. On the lateral aspect of the arm, too, just between the posterior border of the biceps muscle and the anterior portion of the triceps, it is especially superficial and unprotected. The chief causes of injury to this nerve are pressure of the head during sleep, pressure of a crutch, pressure or irritation of callus after fracture of the humerus, and more rarely blows and wounds. The nerve itself is not usually the seat of active inflammation from cold. It may, however, suffer with all the other nerves in multiple neuritis. In alcoholic multiple neuritis, for instance, the branches of this nerve, like all nerves supplying extensor muscles, are especially liable to involvement. In lead palsy the characteristic wrist drop is caused by paralysis of the branches of this nerve in the lower arm and of the muscular branch which supplies the extensor carpi radialis longior. Lead, however, has a peculiar selective action upon the branches of the musculospiral nerve, but this will be described later. Of these causes the most common is undoubtedly the pressure caused during sleep by the patient's head resting upon the humerus. This usually happens only in the deep sleep caused by alcoholic intoxication. In numerous cases of paralysis of the musculospiral nerve observed by me, this cause has always been the active one, with the exception of a few instances in which the paralysis was due to the pressure of a crutch. The explanation for the fact that, as a rule, only drunken men suffer from this paralysis, is that the pain and discomfort caused by prolonged pressure of the head on the arm, which are sufficient to arouse a sober man, or at least to cause him to shift his position, are unheeded by the former. The drunken man is so insensible to this pain that he sleeps on, possibly for many hours, and thus with prolonged pressure paralyzes the nerve. From this circumstance paralysis of the musculospiral

nerve has been called "Sunday morning palsy." I have never seen it occur from pressure of the head during sleep in case of a sober man. The next most important cause of this paralysis is the pressure of the head of a crutch upon the nerve just as it emerges from the axilla. The paralysis may even be bilateral, as in an instance which I saw in a man after amputation of the leg. In this case the patient, who was a large heavy man, was allowed to get up and go about with two rudely made crutches, the heads of which were not properly shaped and padded. As a patient who is obliged to use a crutch is already sufficiently crippled, it is unfortunate that he should be still further disabled by a paralysis of one of the chief nerves in his arm, which could as well be avoided with a little care. The musculospiral nerve has been wounded in various ways (Cheever[108]). Owen[109] reports a case of gunshot injury to the musculospiral nerve, but such cases are exceedingly rare in medical literature.

The symptoms of paralysis of the musculospiral nerve are characteristic and unmistakable. As this nerve is almost exclusively concerned with extension and supination, the abolition of these functions is more or less complete and quite uncomplicated. The exact extent of the paralysis, however, varies according to the point of pressure. If this pressure is within the axilla or just at the emergence of the nerve from the axilla, as when it is caused by the head of a crutch, all the muscles supplied by the nerve, including the triceps, are involved. In the case of drunken men, however, the pressure is usually below the branches of the triceps, and then the paralysis is especially marked in the supinator longus and the extensors of the wrist and fingers. In some few cases, however, the supinator longus escapes. The degree of paralysis varies. When it is complete the patient cannot extend the hand at the wrist, and has much diminished power if not complete absence of power of supination. There is, moreover, some failure in power of flexion of the fingers, as is always the case in paralysis of the extensor muscles. This seems to be due to the fact that the flexors require a certain basis of support for their full activity, which is supplied by the extensor muscles. The loss of power of supination causes a peculiar effort of the patient to compensate by pressing the arm against the side and rotating the humerus outwards. This tends to overcome the extreme pronation of the hand which results when the patient grasps an object. As a rule, sensation is not seriously involved in these pressure palsies of the musculospiral nerve. The upper arm especially escapes. In the lower arm, however, diminished sensibility or even complete anæsthesia may be found, especially on the radial surface of the forearm and on the back of the hand and fin-

gers. Subjective sensations, such as paræsthesia or tingling and prickling, are common for the first few days. This freedom from severe injury of the sensory filaments is what is commonly seen in pressure palsies of mixed nerves.

Muscular atrophy and the reactions of degeneration are not commonly seen in these pressure palsies of the musculospiral nerve. This is due to the fact that in the majority of cases the injury is not severe. The functions of the nerve are abolished only temporarily. The nerve fibres are evidently not seriously impaired, and it is doubtful in fact if their continuity in many of these cases is broken. Exceptions occur, of course, in which all the symptoms of severe injury are observed. In the very rare instances of gunshot or other wounds all the phenomena of complete and perhaps irreparable degeneration of the nerve may follow. This will depend of course upon the severity of the wound.

The *diagnosis* of paralysis of the musculospiral nerve is not difficult. From ordinary lead palsy it is distinguished by the fact that it is usually unilateral, and by the highly characteristic fact that the supinator longus muscle is involved. The history of the case, moreover, is usually very clear. The onset is sudden, due to pressure during drunken sleep or caused by the head of a crutch. In alcoholic neuritis, while the extensor muscles are especially likely to be impaired, the affection is never limited to one nerve trunk.

The *prognosis* of musculospiral paralysis from the causes mentioned is usually favorable. The duration of the case will depend of course upon the severity of the injury. In case the nerve undergoes complete degeneration and the reactions of degeneration with wasting appear in the muscle, the course may be slow and tedious. Many of these cases recover in a few weeks, but the more severe form may last for months.

The *treatment* for pressure palsies of the musculospiral nerve is simple in the extreme. These cases tend to recover, in fact, without active treatment. Electricity to the paralyzed muscles, rest, and massage are usually all that is required. In case the trunk of the musculospiral nerve should be severed by a knife or pistol wound nerve suture should be immediately performed.

Diseases of the Posterior Interosseous Nerve.

This nerve is one of the two terminal branches of the musculospiral nerve. In part of its course it is deeply situated on the interosseous membrane. It supplies all the muscles of the radial and posterior brachial regions, except the anconeus, supinator longus,

and extensor carpi radialis longior. It is consequently the nerve which is most affected in the ordinary form of lead palsy. In this paralysis the muscles affected are especially the extensors of the fingers and of the wrist. As an almost invariable rule, the supinator longus for some unknown reason escapes, and the extensor of the metacarpal bone of the thumb is not completely paralyzed although it may be paretic. The reason for the exemption of these two muscles in lead palsy has not been satisfactorily stated. The supinator longus is not supplied by the interosseous nerve, but by a branch of the main trunk of the musculospiral nerve. The extensor of the metacarpal bone of the thumb, however, is supplied by the interosseous. It seems that the poison in these cases has a selective action and must be expended largely upon the interosseous nerve instead of the main trunk of the musculospiral. This is in accord with the clinical facts, except that the extensor of the metacarpal bone of the thumb escapes, while, on the other hand, the long radial extensor of the wrist, which is supplied from the main trunk of the musculospiral, is usually involved. In cases of lead poisoning, however, it must be recalled that many muscles and nerves in other regions of the body are involved, so there is no positive law limiting the action of the poison to the extensor muscles of the wrist. The type of paralysis just referred to, however, is undoubtedly a very common one in lead poisoning and produces the characteristic wrist drop.

The symptoms of wrist drop due to lead poisoning are as follows: The hand cannot be extended at the wrist in the worst forms. In some cases, however, the hand can be slowly extended with a jerky or spasmodic movement when the fingers are flexed. When the fingers are extended the paralysis is absolute. This seems to depend upon the fact that flexion of the fingers gives the weakened extensors a more advantageous purchase or basis of support. The supinator longus muscle is not involved (see Fig. 31). It stands out prominently, especially when the extensor muscles are atrophied, and presents one of the most characteristic features of lead palsy. The extensor of the metacarpal bone of the thumb is usually not completely paralyzed, although it does not escape entirely. Because of the absolute loss of power in many of the other extensor muscles the movements of the tendon of this muscle may appear quite conspicuous. As the interossei and lumbrical muscles are unaffected the distal phalanges of the fingers may usually be extended, but this function is aided by a passive extension of the proximal phalanges, which are paralyzed.

Lead palsy being due to the action of a poison upon the nerve

trunk has all the characteristics of a peripheral palsy. The muscles atrophy and the reactions of degeneration appear. These reactions in mild cases may not be complete, but in the more severe forms all electro-excitability of the muscles may eventually be lost. The atrophy of the muscle is sometimes extreme. As a rule, sensation is not impaired in lead palsy. The principal sensory branches of the musculospiral nerve are from its main trunk and through its radial division, and the exemption of these demonstrates again the selective action of the poison. In some cases, however, there is dimin-

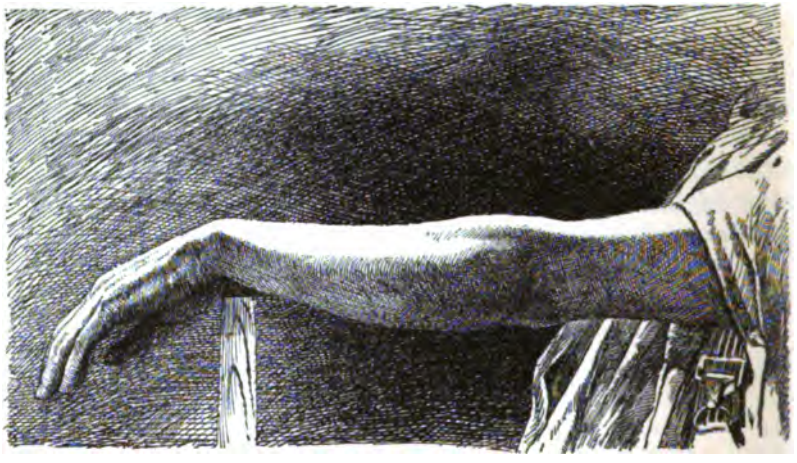

FIG. 31.—Paralysis of the Extensors of the Hand, with Preservation of Size and Power in the Supinator Longus Muscle in a Case of Lead Poisoning.

ished sensibility. This is seen in grave forms of lead palsy in other regions of the body even more markedly than in the forearm. Thus I have seen tactile anæsthesia well marked in the legs in cases of pseudotabes due to multiple neuritis caused by lead. In the forearm, however, tactile anæsthesia is not common in my observation in the ordinary wrist drop caused by lead.

Lead palsy is always bilateral. I know of no instance in which it has been confined to one arm. In some cases, however, one arm is affected before the other or may be worse affected than its fellow. Some observers claim that the right arm suffers sooner and worse than the left. The inference is that this is due to the local absorption of the poison. Manouvriez[110] called attention to the local effects of lead and claimed that these local effects correspond to the local application of lead to the skin. In some of his cases, for instance, the workmen had used their feet to stamp lead and had loss of power in the leg. Anæsthesia has occurred in an area to which

lead had been applied. Pain is not a common symptom in wrist drop due to lead. All forms of subjective sensory symptoms as a rule are wanting.

The diagnosis of lead palsy of the forearm can be made first from the distribution of the palsy itself. The exemption of the supinator longus and of the extensor of the metacarpal bone of the thumb distinguishes it from most cases of paralysis of the musculospiral nerve. The extreme muscular wasting with reactions of degeneration and its slow tendency to recovery in some cases are also rather in contrast with pressure palsy. Second, the history of the case is usually determinative, although it must not be forgotten that lead poisoning sometimes occurs insidiously and from very obscure sources. In all cases, however, there are usually other symptoms of plumbism, the most prominent of which are the blue line on the gums and colic. Third, lead palsy is uniformly bilateral, whereas pressure palsy, as a rule, is unilateral. This rule, however, has exceptions, as in the case above referred to, observed by the writer, in which a man had paralysis of both musculospiral nerves due to the pressure of crutches.

The *prognosis* of lead palsy of the interosseous nerve depends, of course, upon the severity of the lesion. In mild cases under proper treatment and removal of the cause, recovery may take place in from six to eight weeks. In grave cases, however, such as occur in patients who continue to expose themselves to lead after the appearance of their premonitory symptoms, the prognosis is somewhat doubtful. I have known a few cases in which wrist drop with extreme wasting of the muscles had persisted for more than a year and threatened to be permanent. Even the milder cases are rather obstinate and prolonged, and the patient should be told at the beginning that his disability may last for several months.

The *treatment* of lead palsy of the interosseous nerve should be both general and local. A general antidotal treatment should of course be adopted. This is described in detail in Vol. III. of this work. It is sufficient to say that it should consist in the use of the iodides and of sulphuric acid. Of prime importance, however, is the removal of the cause. A patient who has once had lead palsy, or in fact lead poisoning in any form, should never be permitted again to work in lead or to be exposed to it in any way, if this can possibly be avoided. Such patients are especially susceptible to recurring attacks. This is true particularly of the symptoms here under consideration. The local treatment of lead palsy consists in the use of electricity, massage, and strychnine under the skin over the affected muscle. This treatment, it cannot be disguised, does not always

give rapid or brilliant results. It should, however, be persevered in patiently. In cases in which faradic contractility is abolished the galvanic current alone should be used.

Diseases of the Posterior Thoracic Nerve.

This nerve, called also the external respiratory nerve of Bell, arises by two roots from the fifth and sixth cervical nerves just before their junction in the brachial plexus. It has a very long course upon the outer surface of the serratus magnus muscle, extending along the side of the chest to the extreme lower border of that muscle, which it supplies with numerous filaments (Gray). In the neck it penetrates the middle scalenus muscle and it is at this point that it is probably most exposed. This nerve is distributed entirely to the serratus magnus muscle and thus is one of the most important respiratory nerves. The muscle arises by nine heads from the outer surface of the eight upper ribs, the second rib having two, and is inserted into the posterior border of the scapula. Its action, therefore, when the shoulder is fixed, is to elevate the ribs and thus expand the chest. When the chest is fixed this muscle rotates the inferior angle of the scapula forwards, thus elevating the shoulder. It also tends to hold the scapula steadily to the side of the chest. By its action in fixing and elevating the shoulder it assists in carrying weights. These actions must be understood by the clinician to enable him to recognize the rather anomalous appearance caused by paralysis of the serratus magnus muscle.

The posterior thoracic nerve may be injured in a variety of ways. One of the commonest is from carrying a heavy weight on the shoulder. Muscular exertion also seems in some instances to act as a cause. This is probably because the course of the nerve through the scalenus muscle renders it peculiarly liable to compression and in some slight degree to torsion. Thus paralysis of the nerve has been known to follow severe exertion with the upper arm: and it has also occurred during parturition. Blows and wounds about the neck or upon the chest wall may also involve the posterior thoracic nerve. Exposure to cold may possibly act as a cause, and it is possible that some cases, erroneously ascribed to overexertion, may be due to exposure to cold when the surface of the body is overheated and perspiring. Injuries and isolated diseases of the posterior thoracic nerve are rather rare. The serratus magnus muscle, however, may be involved in more widespread degenerative affections, such as progressive muscular atrophy. Paralysis of the respiratory muscles is not uncommon in alcoholic multiple neuritis. Thus the patient may be unable to ex-

pand the chest as much as an inch. In these cases the posterior thoracic nerve is probably the seat of inflammatory changes.

The symptoms of paralysis of the serratus magnus muscle are easily recognized when once understood, but these cases are so rare that the unique appearance caused by this paralysis may puzzle some observers. The striking symptom is a preternatural mobility of the scapula. This is shown especially when the arm is elevated. The scapula then, instead of being held firmly to the side of the chest, as a basis of support for the arm, recedes widely from the chest at its posterior edge, and at the same time rotates so that its inferior angle flies upwards and outwards and the acromium tends to descend. This extreme mobility of the scapula produces two striking effects. First, it gives the patient a peculiar "winged" appearance, caused by the odd movement of the scapula away from the chest wall. This movement leaves a deep sulcus between the posterior edge of the scapula and the chest wall. Second, the hampered movement of the arm when it is elevated, due to a loss of its base of support, is unlike anything caused by other paralyses. The inspiratory movement of the chest on the affected side, especially deep inspiration, is also affected by paralysis of this muscle. The chest is visibly not expanded on the affected side. In some cases pain about the neck is a prominent symptom in the early stages of paralysis of the posterior thoracic nerve. This probably indicates that the nerve is affected by an inflammation of a rheumatic character or is due to cold at the point where it penetrates the scalenus muscle. This cause, I am convinced, is more common than is generally supposed. The paralysis, in the great majority of instances, is unilateral. Cases have occurred, however, in which both nerves were affected. I once saw such a case in a child. The affection is more common in men than in women, and this evidently because the former are more exposed to the causes which produce it—*i.e.*, severe muscular exertion, followed by checking of perspiration due to exposure to cold. In severe cases the muscle is no doubt the seat of the reactions of degeneration, but these can be demonstrated only with great care by applying one pole to the neck over the region of the scalenus muscle and passing the other along the fleshy heads of the serratus magnus muscle where they are attached to the ribs.

The prognosis in these cases should be guarded. As in the case of paralysis of the seventh nerve due to exposure to cold the course of this affection is sometimes tedious. Cases may persist for some months.

In cases in which pain is present over the scalenus muscle active counter-irritation by means of a blister should be used. The salic-

ylates also may be given in cases in which it is suspected that cold has acted as a cause. Electricity, especially the galvanic current, may be used. In severe cases it is well to put the arm at rest even for a while in a sling.

Diseases of the Suprascapular Nerve.

The suprascapular nerve arises from the posterior trunk of the brachial plexus (Van Gehuchten*). It enters the supraspinous fossa through a notch in the upper border of the scapula, passes beneath the supraspinatus muscle and enters the supraspinous fossa in front of the spine of the scapula. It supplies the supraspinatus and infraspinatus muscles. It is occasionally the seat of injury from blows or from carrying heavy weights or, according to Gowers, from luxation of the shoulder-joint, in which latter case the circumflex nerve also may be injured. The paralysis of the supraspinatus and infraspinatus muscles interferes with sundry movements about the shoulder-joint, especially the excursions of the humerus. The act of writing, for instance, may be impaired, the patient being unable to make the lateral movements necessary to move the pen. As the infraspinatous muscle wastes, the posterior part of the deltoid stands out conspicuously by comparison, and this has led some observers to claim that this portion of the deltoid becomes hypertrophied, but this is doubtful. As the supraspinatus assists the deltoid in raising the arm from the side, this motion is slightly impaired by injury of this nerve. The paralysis of the infraspinatus causes an undue rotation at the head of the humerus inwards; and when the arm is raised it tends to fall slightly forwards because of this paralysis.

The *prognosis* of this affection is favorable, although the duration of the paralysis may be prolonged for weeks.

The *treatment* is by rest and electricity and massage to the affected muscles.

Diseases of the Ulnar Nerve.

The ulnar nerve arises from the inner trunk of the brachial plexus in common with one of the branches of origin of the median nerve. It descends on the inner aspect of the upper arm, at first in close proximity to the brachial artery, but in the lower portion of the arm it deviates from that artery, passing more to the back of the upper arm. Its course lies through a groove between the internal condyle and the

* Gray gives its origin from the cord formed by the fifth, sixth, and seventh cervical nerves, but he differs somewhat from Van Gehuchten in his description of the brachial plexus.

olecranon, at which point it is very superficial and in most persons can be readily felt. Passing from this groove behind the inner condyle it enters the forearm between the two heads of the flexor carpi ulnaris muscle. It descends on the ulnar side of the forearm, being placed upon the flexor profundus digitorum muscle. For most of its course in the forearm it is in close proximity to the ulnar artery. At the wrist the nerve crosses the annular ligament and just beyond this ligament divides into its two terminal branches—the superficial and deep palmar (Gray). The ulnar nerve gives off no branches in the upper arm. At the elbow it gives off an articular branch, and lower in its course it gives off muscular and cutaneous branches and another articular branch which supplies the wrist. The muscles supplied by the ulnar nerve are especially the flexor carpi ulnaris and part of the flexor profundus digitorum, the palmaris brevis, the muscles of the little finger, the dorsal and palmar interosseous muscles, two of the lumbricales muscles, the adductor pollicis, and one head of the flexor brevis pollicis. In consequence of this distribution the ulnar nerve is exclusively a flexor nerve. By the flexor carpi ulnaris muscle it presides partially over the flexion of the hand at the wrist. By the flexor profundus digitorum it presides over the flexion of the last phalanges, especially of the little and ring fingers. By the lumbricales, which are accessories to the deep flexor muscle, it still further controls this movement. By the interossei it partially controls the lateral movements of the finger and the extension of the second and third phalanges and flexion of the first. By the adductor pollicis it controls partially the movement of the thumb towards the palm.

By its cutaneous branches it supplies part of the skin of the forearm, especially that over the ulnar aspect, and also the skin covering the ulnar aspect of the hand, the little finger, and the ulnar side of the ring finger.

The ulnar nerve is especially liable to injury and disease. Its very exposed course in some portions of the arm, especially behind the internal condyle and at the wrist, renders it extremely liable to injury in lacerated and incised wounds of the arm and forearm. All sorts of cuts from all sorts of weapons may injure the ulnar nerve. It has been divided by a stab wound from a knife, injured in the lacerated and contused wounds caused by machinery, and even severed by broken glass. I knew an instance of the latter accident in the case of a house painter, who fell through a skylight, severely lacerating his arm and severing the ulnar nerve and artery. Gunshot and pistol-shot wounds have also involved this nerve. It has been injured by prolonged pressure in the case of a glass-blower at his work.

Ballet[111] records an instance of this kind. The ulnar nerve has also been involved in malignant growths. Thus Briddon[112] related a case of sarcoma of the ulnar nerve which ended in amputation of the shoulder-joint and exsection of the scapula. S. Jones[113] reported a case of neuroma, following trauma, of the ulnar nerve, with loss of function. The nerve has sometimes been dislocated with varying effects upon its function. F. J. Lutz[114] reported an instance of habitual dislocation of the left ulnar nerve. Blattmann[115] also contributed to this subject of dislocation of the ulnar nerve as early as 1851. Wharton[116] has reported a case of dislocation of the ulnar nerve at the elbow, and has given a *résumé* of thirteen other cases from the literature. He says that this accident is usually associated with fracture or dislocation, but it may also result from direct violence, from muscular effort, or from violent flexion of the forearm. This latter accident causes laceration of the fascia, which holds the nerve in its groove behind the internal condyle. The symptoms are pain, tingling, and some disability of the elbow, but they usually subside rapidly. Permanent disability is rare. This is likely to follow in those few instances in which neuritis develops. The nerve should be replaced, and, if necessary, kept in place by fixation of the elbow-joint in plaster of Paris. MacCormick fixed the nerve in place with loops of kangaroo tendon passed through the inner margin of the triceps and then around the nerve.

The ulnar nerve has been injured by fracture of the bones of the arm. Thus Panas[117] relates a case of paralysis of the ulnar nerve, showing itself twelve years after a fracture at the elbow. This was supposed to be due to a gradual filling up of the groove behind the internal condyle in which the nerve runs, and to gradual pressure upon the nerve and alteration of its nutrition. This alteration consisted in a thickening and induration of the nerve, simulating a neuroma.

Avezou[118] relates a case of fracture of the humerus, causing a lesion of the ulnar nerve. This resulted in atrophy of the arm, pains in the elbow, and trophic lesions in the hand; the fingers, the hand, the wrist, and the lower part of the forearm were discolored a violet hue. In this case sensibility had almost entirely disappeared from the dorsal aspect of the hand save in the area of the second and third phalanges of the index, middle, and ring fingers.

Next to injury alcoholic multiple neuritis is probably the most frequent cause of impairment of the ulnar nerve, but this occurs, of course, in conjunction with paralysis of many other nerves of the body. Isolated neuritis of the ulnar nerve, due to cold for instance, is probably never seen. I have never seen nor heard of an unequiv-

ocal example of this cause of neuritis, although some cases which are reported of paralysis of the ulnar nerve, due to exposure during sleep and supposed to be favored by pressure, may possibly have been partially due to cold. Pressure palsy of the ulnar nerve, caused during sleep, is not nearly so common as this kind of palsy of the musculospiral nerve. The position of the nerve on the inner aspect of the arm leaves it less exposed than is the musculo-spiral nerve. Still a few examples have been seen. The nerve may be injured, as already said, in cases of fracture at various points of the bones of the arm and forearm and also in dislocation, especially of the elbow-joint. Gowers refers to a case of inflammation of the ulnar nerve following parturition, and Bernhardt referred to a case following typhoid fever. In both these instances pressure was probably a factor in the causation. In prolonged and difficult labor excessive muscular exertion, with pressure or probably a blow or contusion unconsciously inflicted by the patient herself, might cause injury and paralysis of the nerve.

The *symptoms* of disease and injury of the ulnar nerve are paralysis of the muscles supplied by the nerve, causing a characteristic impairment and deformity of the forearm and hand, anæsthesia in a well-defined territory, and trophic lesions.

The paralysis of the flexor carpi ulnaris muscle, which is one of the two important flexors of the wrist, causes a deviation of the hand towards the radial side on attempts at flexion. This impairs very much the patient's ability to grasp objects firmly and draw them towards him. Flexion of the little and ring fingers is especially impaired; in fact, the little finger is almost entirely paralyzed. The power of adduction of the thumb towards the palm is lost; and the abolition of this most important function seriously impairs the patient's ability to grasp small objects. The paralysis of the interossei and lumbricales muscles causes abolition of the power of flexion of the first phalanges and of extension of the second and third phalanges; but this loss is less in the first two fingers because their muscles are supplied by the median nerve. This paralysis of the interossei and lumbricales muscles causes a very characteristic deformity, which cannot be mistaken after it is once seen. Thus the first phalanges become overextended, much more than is possible by voluntary power, and the second and third phalanges become strongly flexed. This gives the hand a peculiar claw-like appearance, which has been called by the French the "main-en-griffe." This is seen only as a result of paralysis of the muscles supplied by the ulnar nerve, but is rather more complete in cases of muscular atrophy than of peripheral lesions of the nerve trunk, for the reason that

in the former case the lumbricales of the first and second fingers are also involved. In long-standing cases, in which muscular atrophy has occurred, the hand is peculiarly wasted and distorted. The spaces between the metacarpal bones are hollowed out because of the wasting of the interossei muscles. The palm of the hand is especially wasted and hollowed and the hypothenar eminence is flattened. The unaffected thenar muscles stand out conspicuously and the opponens muscle of the thumb causes rotation forwards of that member. Sensation is variously affected in lesions of the ulnar nerve according to their severity. Tingling and burning sensations are not uncommon and may be aggravated by pressure on the nerve behind the internal condyle. As in the cases of all nerves, however, the motor fibres may be more involved than the sensory. This may be so in cases especially of injury caused by pressure. In totally destructive lesions anæsthesia in the area of distribution of the nerve will be complete. This area is on the ulnar side of the hand, in both the palmar and dorsal aspect, and includes the whole of the little finger and the inner half of the ring finger. The anæsthesia may also extend some distance above the wrist on the ulnar aspect of the arm. In irritative lesions of the nerve, such as lacerated wounds and gunshot and pistol-shot wounds, trophic lesions are not uncommon. Thus a portion of the hand and forearm may become swollen and œdematous, and the skin over the ulnar distribution may present the peculiar glossy appearance seen in this kind of nerve injury. These phenomena are accompanied with burning pain, the "causalgia" of Weir Mitchell. In addition to the extreme muscular atrophy other trophic lesions may appear. Herpetic and pemphigoid eruptions may occur and these may break down in ulcers. Occasionally even whitlow may supervene.

The *diagnosis* of lesions of the ulnar nerve is comparatively easy. The symptoms just described are so characteristic as to be unmistakable, and usually the history of the case is clear. In mild cases due to pressure, which has occurred unconsciously to the patient, as during sleep or in the agony of parturition, some little care may be necessary to establish the diagnosis. Even in these cases, however, the impairment of power is quite characteristic. The awkwardness of the patient in flexing the hand, the impairment of ability to hold objects firmly, the weakness of the thumb due to the paralysis of its adductor muscle, the paralysis of the little finger, and the impaired power of flexion of the first phalanges, all constitute a picture which is unmistakable. That the affection is peripheral and confined to the muscles supplied by the one nerve can readily be determined by the preservation of power of all the other muscles in the forearm.

As in all cases of lesions of nerve trunks the muscles degenerate and the reactions of degeneration are usually promptly established. Exceptions may occur in cases of very mild injury, and in such instances electro-excitability of the muscles may not be radically altered. Even in such cases, however, there is usually some diminution or even complete abolition of the faradic contractility.

The *treatment* of lesions of the ulnar nerve will depend of course largely upon their nature and extent. In grave surgical lesions the treatment of the nerve forms part of the general treatment of the case. After the injury to the soft parts has been properly treated the arm should be put at perfect rest. Later, when the wounds have healed, if paralysis of the muscles persists, electricity should be used. In cases in which the nerve has been badly lacerated direct surgical treatment of the wound may be necessary. This is so especially if the nerve has been completely severed. In such a case its ends should be carefully trimmed and sutured with all antiseptic precautions. In long-standing cases, in which muscular atrophy and contracture have occurred, great care and patience will be necessary. The fingers should be supported on a splint, electricity should be applied, gentle massage should be used, and strychnine should be given hypodermically. In cases of slight injury about the olecranon, in which the nerve trunk can be felt to be swollen and sensitive, counter-irritation by means of a blister should be used. Iodide of potassium and the salicylates are of no use in the great majority of cases of injury to the ulnar nerve. In any rare instance, however, in which a rheumatoid inflammation seems to have been excited in the nerve trunk, the patient might have the doubtful benefit of treatment with the salicylates.

The *prognosis* in all cases of injury to the ulnar nerve should be most guarded. In cases of severe injury the progress will be slow and tedious. The resulting deformity will be very great, and most disabling to the patient. In some cases, in fact, permanent disability will result. In cases in which the muscular atrophy has advanced to such an extreme degree that the characteristic "main-en-griffe" has resulted, the prospect for a complete recovery is exceedingly doubtful. Even in such cases, however, persistent efforts should be made with electricity, massage, and passive movements to overcome the deformity and to restore some function to the disabled hand. The importance of this can be recognized when it is understood that the deformity caused by hopeless paralysis of the ulnar nerve entails upon the patient a disability which is even worse than that caused by an amputation of the forearm.

Diseases of the Median Nerve.

The median nerve arises by two branches, one from the inner and one from the outer cord of the brachial plexus. These branches embrace the axillary artery, uniting just in front of or a little to the outer side of that vessel (Gray). Its relations as it passes down the arm are especially close to the artery. At the bend of the elbow it lies on the inner side of this vessel and in close proximity to it. It descends through the middle of the forearm on its anterior aspect just beneath the flexor sublimis muscle. Before reaching the annular ligament at the wrist it becomes much more superficial, lying between the flexor sublimis and flexor carpi radialis and covered only by the skin and fascia. It passes under the annular ligament into the palm of the hand, where it divides into digital branches to the thumb, index, middle, and ring fingers. The median is a mixed nerve, *i.e.*, it contains both motor and sensory fibres. All its branches are given off in the forearm. It supplies all the muscles on the front of the forearm except the flexor carpi ulnaris and the inner half of the flexor profundus digitorum. The muscles supplied by the median nerves, in other words, are largely flexor and pronator, while in the palm of the hand it supplies the abductor, the opponens, and the short flexor of the thumb. It supplies the first and second lumbrical muscles. The median nerve is thus the fellow of the ulnar nerve, both of them being almost entirely flexor.

The sensory fibres of the median nerve supply the palm of the hand and the palmar surfaces of the thumb, index and middle fingers, and the radial side of the ring finger.

The median, like the ulnar nerve, may be the seat of either injury or disease. It is apparently, however, not so often injured as the ulnar or musculospiral nerves. The reason for this must be its location, although it is apparently as much exposed as the ulnar. In the forearm, however, it is rather more protected, and also where it crosses the elbow, as it is there on the internal aspect of the arm, whereas the ulnar crosses that joint in a very superficial location between the olecranon and internal condyle. In cases in which it is injured by incised, lacerated, or contused wounds, there is also of necessity more or less injury to blood-vessels and tendons. The median nerve may be injured and even entirely divided by incised wounds. Boeckel[118] relates such an accident to the median nerve, associated with division of the flexor tendons and the brachial artery. Duret[119] reports a case of contused wound of this nerve, followed by trophic lesions. Ferret[120] reports a case of injury to the median

nerve caused by a surgical operation to the elbow. Lefort [111] relates a case of gunshot wound of the median nerve. Gunshot wounds of the nerve, in fact, seem to be the commonest injury. Lawrie [112] and Warren [113] have related instances of such wounds. Avezou,[114] in his monograph on contusions of the nerves of the arm, has related a number of instances of injury to the median nerve. Isolated diseases of the median nerve, such as inflammation due to cold, are exceedingly uncommon. This nerve is, of course, involved with many other nerves in multiple neuritis from various causes. Thus in the neuritis caused by alcohol the median nerve may be involved, although the muscles supplied by it are not so much affected in this disease as are the extensor muscles of the hand supplied by the musculospiral nerve. Potain [114] has reported a case of paralysis of the median nerve, which he describes as of rheumatic origin. Such a case might possibly be caused by exposure to cold, but this, as said above, must be extremely rare. The nerve is not often involved by pressure of the head during sleep, as is the musculospiral. This is evidently because its position does not favor such an accident. Fractures of the bones of the forearm may involve the median nerve, and redundant callus may make pressure upon it. Violent muscular contraction of the pronator teres muscle has been known to cause paralysis of this nerve (Weber, quoted by Gowers).

The *symptoms* of paralysis of the median nerve are loss of power of pronation and flexion. The forearm cannot be pronated beyond the mid-position, and when this is attempted the patient endeavors to supplement the movement by rotating the humerus inwards and at the same time abducting the elbow from the side. Flexion of the wrist can be accomplished only by the flexor carpi ulnaris. Consequently the hand is strongly flexed towards the ulnar side only. The thumb cannot be flexed and abducted, and, because of the paralysis of the opponens muscle, cannot be opposed to the tips of the fingers. This is a very characteristic disability caused by paralysis of the median nerve. Flexion of the phalanges is lost except for the distal phalanges of the ring and little fingers which are supplied by the ulnar half of the flexor profundus digitorum. The unopposed extensor action of the interossei muscles may cause a subluxation of the articulation between the middle and distal phalanges. The thenar eminence wastes. Anæsthesia, if present, is on the surface of the palm, on the palmar surfaces of the thumb, index, and middle fingers, on the radial side of the ring finger, and on the dorsal tips of the thumb and first three fingers (Fig. 32).

As in all cases of injuries to nerves trophic lesions are not uncommon. In a case reported by Duret [115] such lesions were found. Scars

were formed at the extremities of the fingers as a result of trophic ulceration. Richelot[125] relates a case of wound of the median nerve with adherent cicatrix and anæsthesia and trophic disorders. Voituriez[126] also relates a case of wound of this nerve causing trophic lesions.

The *diagnosis* of paralyis of the median nerve is usually determined by the history of the case and the characteristic distribution of the paralysis of the muscles. The loss of power of pronation, the

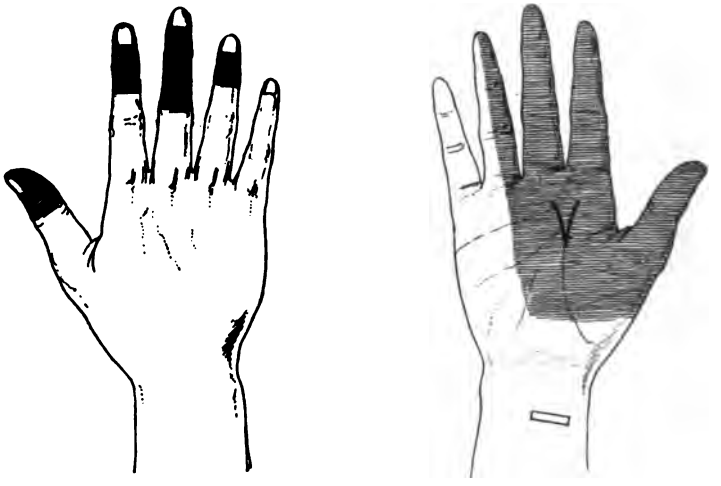

FIG. 82.—Anæsthesia from Paralysis of the Median Nerve. (Avesou.)

awkward flexion of the wrist, the adduction and extension of the thumb, and the loss of power of flexion of the fingers are all characteristic. The affected muscles waste and present the reactions of degeneration, and these symptoms serve to distinguish the paralysis from that caused by cerebral lesions.

The *treatment* of injuries to the median nerve is similar to that indicated for the ulnar. In case of division of the nerve the ends should be freshly pared and sutured. The associated wound of the soft parts will of course require special surgical treatment and the arm should be put carefully at rest. Later, if paralysis persists, electricity, massage, and strychnine should be used.

The distribution of the sensory terminals of the median nerve in the skin is a subject of some interest. Cruveilhier taught that the skin of the dorsal aspect of the fingers received all its sensory supply from the radial and ulnar nerves. Anatomists generally, however, have claimed that the various terminals of the nerves of the arm anastomose, but such anastomosis we recognize now with our

present knowledge of the neuron to be impossible. Létiévant noted later that the facts of pathology were in opposition to the teachings of the anatomists. Richelot collected many observations upon the affections of sensibility occurring after section of the median nerve. According to his observations the dorsal aspect of the last two phalanges of the index and middle finger and the radial half of the last two phalanges of the ring finger receive their sensibility from the median nerve. It is possible also that the dorsal aspect of the last phalanx of the thumb receives its sensory supply from the median nerve. Thus in a case recorded by Durham,[127] of paralysis of the median nerve after wound at the wrist, there was diminution but not abolition of sensibility upon the dorsal aspect of the second phalanx and thumb. Three months after the accident the patient had almost normal movements at the wrist, and could flex and extend his fingers, but not perfectly. The sensibility in the distribution of the median nerve was almost entirely lost, *i.e.*, upon the palmar surface, in the usual area. On the dorsal aspect of the hand sensibility was abolished over the terminal phalanges of the ring, middle, and index fingers, and was diminished but not entirely lost over the terminal phalanx of the thumb. It is possible, however, as Avezou[11] states, that this was an anomalous distribution. It seems in fact that the median nerve presents varieties of distribution, but the one shown in the diagram after Avezou (Fig. 32) probably represents its widest distribution and corresponds with the affected area in Durham's case.

Diseases of the Anterior Thoracic Nerves.

These nerves, two in number, supply the pectoralis major and minor muscles. The external nerve arises from the outer cord of the brachial plexus and is distributed to the under surface of the pectoralis major muscle. The internal nerve arises from the inner cord of the brachial plexus and is distributed to the under surface of the the pectoralis minor and pectoralis major muscles. These nerves may be involved in any lesion high in the brachial plexus. As they arise, however, from the upper part of the outer and inner cords respectively they escape in any lesion of the brachial plexus below the clavicle. Thus in Raymond's case of injury to the retroclavicular portion of the brachial plexus by a large blood clot, causing pressure, these nerves were not involved, consequently the pectoral muscles were not paralyzed. Isolated paralyses of these nerves must be extremely rare. I do not know of any example. They would not likely be injured by any lesion that did not also involve some other branches or some trunk of the plexus itself. In cases of general

muscular atrophy involving the muscles of the shoulder and arm the muscles supplied by these nerves may be wasted. The nerves themselves are never the seat of an isolated neuritis caused by cold or by any poison circulating in the blood. A few instances are on record of congenital absence of the pectoralis major and minor muscles. The exact cause for such an abnormality is not clear. Defective development or absence of these muscles has been attributed to traumatism or to infantile paralysis. Congenital absence of other

FIG. 33.—Absence of the Right Pectoralis Major and Minor Muscles.

muscles has also been noted, although from the literature it seems that this absence of the two pectoral muscles is rather the most common.

The following case of congenital absence of the pectoralis major and minor muscles is recorded here as an instance of this rather rare anomaly, although it has been impossible to associate it with injury or disease of the anterior thoracic nerves.

P. M——, aged 53, white, was admitted into the Philadelphia Hospital, under my care, for syphilitic meningomyelitis of the middorsal region. The muscular anomaly is as follows: On the right side of the chest there is great depression and flattening in the region of the pectoral muscles. Examination shows that there is

complete absence of the lower belly or sternal portion of the greater pectoral muscle and of a portion of the upper belly or clavicular portion. Only that small part of the muscle remains that is attached to the middle third of the clavicle. The pectoralis minor muscle is entirely absent. Upon abduction of the arm the costocoracoid membrane stands out prominently as a fibrous band, and the small remaining portion of the clavicular belly of the pectoralis major becomes knotted and firm, showing good development. In no movement of the arm has the patient noted the least weakness or inconvenience, and this is a remarkable feature of the case. He says he is as strong in his right arm as in his left. He has always been used to hard work as a laborer, most of his time having been spent with the pick and shovel in a stone quarry. The asymmetry of the right side of his chest, he says, has been present since birth. No history of dystocia could be obtained. There is no history of malformation in other members of the family. The only impairment of motion is seen when the patient's arm is forcibly abducted and held, then when he is bidden to adduct the arm across the chest there is evidently some failure of power, which is made up in part by violent movements of the deltoid muscle.

Azam and Casteret[128] have reported the cases of two young soldiers in whom the pectoral muscles were absent. In one case the pectoralis major was reduced to a slender fasciculus composed of the clavicular portion, and to an hypertrophied fasciculus attached to two ribs and to the border of the sternum. The pectoralis minor was a slender fasciculus merely. In the second case the pectoral muscles on the left side were entirely wanting.

Féré[129] gives a picture of an epileptic boy with congenital absence of the great pectoralis muscle of the left side. He says it is the only anomaly of the kind he has seen in an epileptic. No significance, however, can be attached to the anomaly with respect to epilepsy. Poland[130] reported a case of absence of the pectoral muscles in a convict.

Hofmann[131] reports a case of a man aged forty-seven who presented the following condition: The sternal portion of the right pectoralis major was completely absent, while the clavicular portion and the pectoralis minor were well developed. The entire upper limb was shorter and smaller than its fellow. The fingers of the right hand were in the position of the claw hand. Between the right index and middle fingers and between the latter and the ring finger there was a bridge of skin to the middle of the first phalanx. Between the ring and little fingers there was a similar bridge of skin extending a little farther forwards. On the volar side of the right hand the interphalangeal furrows were absent on all the fingers but not on the thumb. In spite of the atrophy the right arm was as powerful as the left. Hofmann says that only ten cases of deficiency of the breast

muscles with this condition of the hand have been reported. In most of these cases the patients were men and the anomaly was on the right side.

Diseases of the Musculocutaneous Nerve.

This nerve arises from the outer cord of the brachial plexus opposite the lower border of the pectoralis minor muscle. After perforating the coracobrachialis muscle and the deep fascia it becomes cutaneous. It supplies the coracobrachialis, biceps, and brachialis anticus muscles. It also supplies some sensory filaments to the skin of the ball of the thumb, and by a posterior branch it supplies the skin of the lower third of the forearm on the radial aspect (Gray).

The musculocutaneous nerve, so far as I know, is never paralyzed alone. It is involved, of course, in lesions of the brachial plexus, in which case there is paralysis of the muscles supplied by it, *i.e.*, the biceps and brachialis anticus. As these muscles flex the arm upon the forearm the power to effect this movement is lost. The paralysis also causes anæsthesia on the radial aspect of the forearm, both front and back.

Diseases of the Internal Cutaneous Nerve.

This nerve arises from the inner cord of the brachial plexus near the origin of the ulnar nerve. It supplies the skin over the biceps muscle and over the ulnar aspect of the forearm as far as the wrist, both front and back.

This nerve is never paralyzed alone. It suffers, of course, in lesions of the brachial plexus, in which cases there is anæsthesia in its area of distribution, caused by interruption of its fibres.

Diseases of the Dorsal and Intercostal Nerves.

There are twelve pairs of dorsal nerves. As their name implies, they arise from the dorsal portion of the spinal cord and pass out through the intervertebral foramina, one beneath each dorsal vertebra. These nerves are rather small in size compared with the cervical and lumbar roots. Their intraspinal course (*i.e*, their course from their origin in the cord to their point of emergence through the intervertebral foramina) is rather longer than in the case of the cervical nerves. At their exit from the intervertebral foramina these nerves divide into two branches: a posterior branch or dorsal nerve proper, and an anterior branch, usually called intercostal nerve.

The dorsal branches supply the muscles of the back and give off cutaneous branches which supply the skin with sensory filaments.

The anterior branches of the dorsal nerves (called usually the intercostal nerves) are distributed to the walls of the chest and abdomen. They are entirely separate from each other—*i.e.*, they are not joined together in a plexus, in this respect differing from the other groups of spinal nerves. Each nerve has a connection with a ganglion of the sympathetic system. These nerves pass forwards in the intercostal spaces. In part of their course they lie in close proximity to the pleura. The members of the upper group, or first six intercostal nerves, have a slightly different course from the lower six of the second group, as is pointed out by Gray; but this distinction is not of much clinical importance. The first group supply the skin of the mammæ and front of the chest, and give muscular filaments to the intercostal and triangularis sterni muscles. The principal branches of the intercostal nerves are the lateral cutaneous nerves, which arise midway between the spine and the sternum. These are cutaneous branches largely supplying sensory filaments. The lower group of six intercostal nerves supply the rectus muscle and the skin of the front of the belly as well as intercostal and abdominal muscles. They supply the skin especially of the sides of the abdomen and back.

The first and last dorsal nerves have some peculiarities.

The first has resemblances to the cervical nerves, especially in the mode of distribution of its dorsal branch. Its anterior branch forms part of the brachial plexus. It has a small intercostal nerve which, however, gives off no lateral cutaneous branch.

The last dorsal nerve is larger than the others. It has communications with the iliohypogastric branch of the lumbar plexus and sometimes with the first lumbar nerve. Its lateral cutaneous branch is a very large nerve and is distributed to the skin of the front of the hip (Gray).

The dorsal and especially the intercostal nerves may be the seat of various lesions, and in fact not infrequently furnish important indications for diagnosis. The roots of these nerves are frequently involved in meningitis of the spinal cord. They may also be injured, inflamed, or pressed upon in spinal caries, and under these circumstances, especially if only one or two nerve trunks are involved, they present most misleading symptoms. Obelinsky'" reports the case of a man with a syphilitic history dating back twenty years, who had violent intercostal neuralgia for several years. Treatment had failed. The case was one probably of localized syphilitic leptomeningitis, irritating a nerve root, and this diagnosis was apparently confirmed by the fact that the affection was cured eventually with hypodermic injections of calomel. In syphilitic leptomeningitis early and impor-

tant indications are sometimes furnished by irritation of the roots of the dorsal nerves. Probably syphilis of the meninges and tuberculous caries of the bones are the two most common causes of symptoms in the course of the intercostal nerves. Tumors of the spinal cord often cause irritation of one or more roots of the dorsal nerves, and this symptom may closely simulate simple intercostal neuralgia. The causes just enumerated are of course central or almost central in their location, but when it is considered that the intraspinal course of the dorsal nerves is a long one and that therefore they are especially liable to injury from lesions within the spinal canal or in the bones of the spine, it is seen that these are properly instances of peripheral lesions; for it matters not whether a nerve trunk be injured within or without the spinal canal, the lesion is practically in either case peripheral. A common cause of disease of the intercostal nerves or possibly of the posterior ganglia of the dorsal nerve roots is the poison of herpes zoster. The chest is frequently invaded by this disease and its location is determined by the course of the intercostal nerves. The chest in fact is rather more commonly attacked than is any other region. The intercostal nerves are frequently the seat of severe neuralgia. This probably is the expression of slight nutritive changes in the sensory neurons, caused by cold or by the action of some poison in the blood. These nerves may also be injured by wounds, bruises, and fractures. Perforating wounds of the chest may possibly involve one or other of the intercostal nerves. Such instances, however, are uncommon, as the nerve is well protected by the bone. In fractures of the ribs, however, one or more of the intercostal nerves may possibly be injured. In case of vicious union, deformity, and redundant callus, irritation of one of these nerves may become chronic and cause more or less persistent neuralgic symptoms.

In alcoholic multiple neuritis it is possible that these nerves may be involved, especially their motor branches to the intercostal muscles. In this disease paralysis of the movements of the chest in respiration is not uncommon, especially in the graver form. I have occasionally seen the power of expansion of the chest almost entirely abolished. This constitutes a grave complication.

The *symptoms* of lesions of the dorsal and intercostal nerves are both sensory and motor. The sensory symptoms are usually pain and occasionally anæsthesia. In cases of meningitis in which a few nerve roots in the dorsal region are involved it is not uncommon to observe what is known as the "girdle symptom." This consists of a sense of constriction about the chest, waist, or abdomen, which is frequently described by the patient as a feeling as though a string

or band were tied tightly about the body. This is seen not uncommonly in locomotor ataxia and more commonly still in syphilitic meningomyelitis, especially that form of the disease which is known as "Erb's palsy." Acute localized pain in one intercostal nerve has been observed in the early stages of spinal caries. This may closely simulate and has been mistaken for an attack of ordinary intercostal neuralgia. It may or may not be unilateral. It is usually associated with some spasmodic action of the muscles of the back, hence with stiffness of the back and with restriction of the free movements of the trunk. Such movements, as well as pressure over the vertebra and jars and shocks, such as may be produced by a blow on the top of the head or by the patient jumping off a low stool, usually increase this pain. Close inspection will usually reveal commencing deformity. Pain along the course of one or more dorsal or intercostal nerves is not uncommon in cases of tumor of the spinal cord in the dorsal region. In spinal tumors the pains are more likely to be eccentric, because of the limited character of the lesion and the almost exclusively peripheral distribution of the nerves (Mills and Lloyd [133]). In these cases also, as Leyden pointed out, movements of the spinal column are often difficult and painful. As in the case of spinal caries there is also stiffness of the back and trunk. Girdle sensations are not uncommon in cases of tumor of the dorsal cord. According to Mills and Lloyd, tumors located in the dorsal region give this sensation not only in the chest but also in the abdomen, the reason for which can be understood by reference to the anatomy of the parts. The localizing value of this sensation is not always exact. Thus in a case reported by Pel [134] a tumor at the level of the sixth and seventh cervical vertebræ caused a constriction sense about the legs and abdomen, and according to Lachmann [135] a glioma of the filum terminale caused a constriction sense of the chest, but this latter observation is open to doubt. In cases of tumor of the spinal cord paralytic symptoms are sooner or later associated with the disorder of sensation. In herpes zoster the disease usually begins with neuralgic pains, which are felt especially along the course of one or two intercostal nerves. These pains usually precede the eruption by several days. They are of a very severe character and are attended with heat and burning sensations in the skin. The appearance of the eruption and its peculiar characteristic scon indicate the true nature of the lesion. The disease is unilateral and depends upon an inflammation of the sensory neurons in the intercostal nerves and probably more especially of their cell bodies in the posterior ganglia. In most of these severe forms of involvement of the roots of the dorsal nerves or the trunks of the intercostal nerves

there is some embarrassment of respiration. This is probably not truly paralytic, but depends upon the increased pain which is caused by efforts to take a deep inspiration. Simple intercostal neuralgia is marked by pain along the course of one or more intercostal nerves. It may also cause some inhibition of respiration. It may closely simulate pleurisy, but can be differentiated by the absence of the physical signs of the latter. In hysteria a peculiar form of neuralgic pain, centred about the mammary gland, especially in women, is sometimes seen. This is called *mastodynia*. It is usually associated with other hysterical symptoms. The patients in these cases are usually the victims of the dread of cancer of the breast and are otherwise neurotic.

I have known one instance in which an aneurysm of the thoracic aorta caused severe localized pain along the course of one of the intercostal nerves.

The *diagnosis* of affections of the intercostal nerves often requires great care. In case of grave organic lesions, such as spinal caries, syphilitic meningomyelitis, and tumor of the spinal cord, this intercostal pain may be misinterpreted, especially in the early stages.

In cases of spinal caries the diagnosis will be assisted by the spasm of the dorsal muscles, restriction of the movements of the chest, increased pain on movement and from shocks and jars, and by the commencing deformity of the vertebræ.

In cases of tumor of the spinal cord the earliest symptom is sometimes irritation along one nerve trunk. Later, however, paralytic phenomena supervene, such as paraplegia, more marked possibly in one leg, loss of control of the bladder, and various areas of anæsthesia.

In syphilitic leptomeningitis the girdle sense is sometimes an early phenomenon, but other symptoms are soon apparent. Thus in a case of a young man, recently seen by me, girdle sense was well marked and was attributed by the patient to his having lifted a heavy weight. He had abolished knee jerks, however, and some impairment of his gait.

Herpes zoster is always heralded by severe neuralgic pains along the course of one or more intercostal nerves. The eruption, however, makes its appearance in a few days and determines the diagnosis. In simple intercostal neuralgia the disease can be distinguished from pleurisy by the absence of fever and the physical signs. In hysterical mastodynia the peculiar localization of the pain and the patient's mental and other hysterical symptoms make the diagnosis comparatively easy. In the case of aneurysm of the aorta, above referred to, the symptoms were so strongly suggestive of a focal lesion, apparently tumor, of the spinal cord, that a consultation was called

for the purpose of determining the propriety of an operation. In this case only the most careful and expert physical examination finally determined the presence of an aneurysm.

The *treatment* of the various affections of the dorsal and intercostal nerves, just enumerated, depends of course upon their cause. Tumors of the spinal cord do not yield to drugs. The treatment, if successful, must be surgical. The same may be said of spinal caries. The disease falls to the care of the orthopedic surgeon, and the treatment is conducted especially with rest and with the aid of suitable braces and jackets. In the early stages of syphilitic leptomeningitis of the spinal cord a most energetic antisyphilitic treatment is indicated and will sometimes be followed with success. In the case of Obelinsky, above referred to, cure was affected by the hypodermic injection of a mercurial. This mode of administering mercury in the early stages of nerve syphilis cannot be too highly recommended. The treatment may be supplemented with large doses of iodide of potassium. Counter-irritation, as with blisters, and even with a hot iron, is recommended by some, and has seemed in some cases to be useful, but it can be regarded only as an adjuvant to thorough constitutional treatment. In simple intercostal neuralgia treatment with the salicylates and with some of the coal-tar derivatives, such as phenacetin, antipyrin, etc., is indicated. Counter-irritation in these cases sometimes does good. The treatment of herpes zoster has been conducted with a large variety of drugs. This treatment is best described in works on dermatology. In hysterical mastodynia suggestive therapeutics is largely indicated. The danger from the use of sedative drugs, especially morphine, is that the patient may form some drug habit.

Diseases of the Lumbar Plexus.

There are five pairs of lumbar nerves. They emerge from the foramina, one beneath each lumbar vertebra. The roots of the lumbar nerves (*i.e.*, the portions of these nerve trunks within the spinal canal) are much longer than those of the cervical nerves. This is due to the fact that the spinal cord terminates opposite the second lumbar vertebra, and that consequently the roots of the lumbar nerves must traverse some space before reaching their respective foramina. These nerve roots thus go to form in part the leash of nerves known as the cauda equina. These lumbar nerve roots are the largest of all the spinal nerves. As in the case of all the nerves these roots do not become joined until they reach the intervertebral foramina. The posterior are rather larger than the anterior. At their exit from the foramina the lumbar nerves divide into an anterior and a posterior

branch. The posterior branches supply the erector spinæ, the interspinales and some other muscles of the back, and also the skin of the gluteal region. The anterior branches of these lumbar nerves, after communicating with the ganglia of the sympathetic system, pass outwards behind the psoas magnus muscle. The branches of the four upper nerves are joined together by connecting branches and form the lumbar plexus. The anterior branch of the fifth lumbar nerve, after uniting with a branch from the fourth, passes downwards across the sacrum to unite with the anterior branch of the first sacral nerve, and thus goes to assist in the formation of the sacral plexus. The lumbosacral nerve is the cord that results from the union of these two nerves.

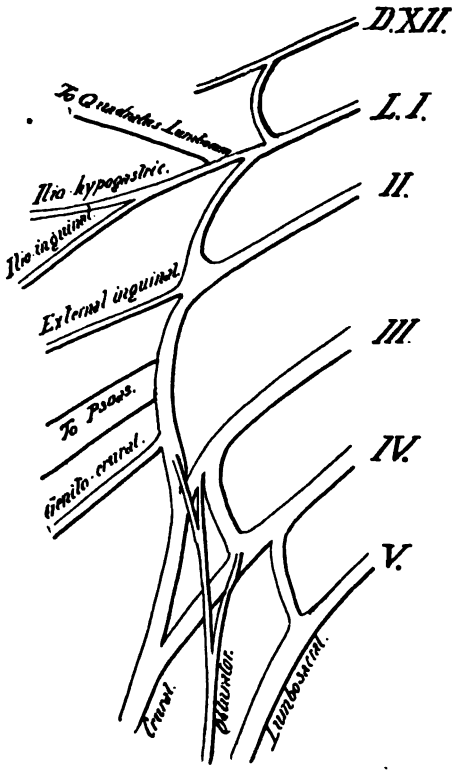

FIG. 34.—Diagram of the Lumbar Plexus.

The lumbar plexus, formed from the union of the twelfth dorsal and the four lumbar nerves, is more simple than the brachial plexus and may be described as follows: The plexus is located in front of the transverse processes of the lumbar vertebræ. These various nerves are united by loops. Thus the twelfth dorsal sends a loop to the first lumbar and this in turn sends a loop or branch to the second lumbar. The first lumbar nerve, after sending its branch to the second lumbar nerve, divides into the iliohypogastric and the ilioinguinal nerves. The second lumbar nerve, after receiving its branch or loop from the first lumbar nerve, gives off the external and internal inguinal nerves and then unites with the third lumbar nerve. The third lumbar nerve, after sending a branch or root to the fourth lumbar nerve, unites with the second and this trunk unites with the fourth to form the crural nerve. The obturator nerve arises from three roots springing respectively from the second, third, and

fourth lumbar nerves. The fifth lumbar nerve has no connection with the other lumbar nerves except by the loop which it receives from the fourth. It goes to form the lumbosacral cord, already referred to, and forms in fact a part of the sacral plexus. The principal branches of the lumbar plexus are the iliohypogastric and the ilioinguinal, the external and internal inguinal, the crural and the obturator, and the lumbosacral nerves (Gray, Van Gehuchten).

The iliohypogastric nerve arises from the first lumbar nerve. It is a sensory nerve and is distributed to the skin of the gluteal and hypogastric regions.

The ilioinguinal nerve arises from the first lumbar nerve in common with the iliohypogastric. It supplies motor filaments to the internal oblique muscle. It emerges at the external abdominal ring in company with the spermatic cord and is distributed to the skin of the scrotum in the male or to the labium in the female and to the upper and inner part of the thigh. The genitocrural nerve arises from the second lumbar. Its genital branch supplies the cremaster muscle in the male. In the female it is lost in the round ligament. Its crural branch is distributed to the skin of the upper anterior aspect of the thigh.

The external cutaneous nerve, called also the external inguinal, arises from the second lumbar. It is a sensory nerve largely and is distributed to the skin along the anterior and outer part of the thigh as far down as the knee, while its posterior branch supplies the posterior surface of the thigh as far as its middle third.

The obturator nerve arises by three branches (Van Gehuchten)—or two branches, according to Gray—from the second, third, and fourth lumbar nerves. It supplies the obturator externus and the adductor muscles of the thigh. It sends an articular branch also to the hip joint. This nerve is largely motor and is concerned in adducting the thigh.

The anterior crural nerve is the largest branch of the lumbar plexus. It supplies all the muscles of the front of the thigh except the tensor vaginæ femoris, and supplies sensory filaments to the front and inner side of the thigh and to the leg and foot and also articular branches to the knee. It is formed by the junction of the second, third, and fourth lumbar nerves, but chiefly from the latter two. It passes into the thigh beneath Poupart's ligament, on the outer side of the femoral artery. Its longest branch is the internal saphenous nerve. This branch supplies sensory filaments to the front and inner side of the leg. It is thus seen that the lumbar plexus supplies especially the adductor muscles and the extensor muscles of the thigh and

leg, and gives sensory filaments to the anterior and interior surface of the thigh and leg.

The lumbar plexus is much less frequently affected by disease and injury than is the brachial plexus. Meningitis in the lumbar region of the cord and of the cauda equina may affect the nerve roots. Such meningitis may be caused by syphilis, although syphilitic disease is rather less common in the lumbar than in the dorsal region. These nerve roots are also involved in injuries to the cauda equina, which will be described later. Such injuries are caused by falls, especially when the patient lands upon the buttocks, the mechanism being sometimes by hemorrhage into the lower part of the spinal canal. Caries of the bones of the lumbar vertebra may also cause irritation or paralysis of some of these roots. Abscesses within the sheath of the psoas muscle may involve some of the branches of the lumbar plexus. The plexus itself is not usually involved in parturition, but its two main branches—the obturator and the anterior crural nerves—may be injured in this process. Luxation of the hip-joint may injure the anterior crural nerve. Tumors in the abdominal cavity may cause pressure upon this plexus, the symptoms depending of course upon the severity of the lesion and the exact nerve trunks involved. Most of the symptoms arising from injury to the lumbar plexus, however, will best be described with reference to the individual nerve trunks implicated, and the appropriate treatment can then be indicated.

Before proceeding, however, to a consideration of the symptoms of individual nerves it will be well to consider briefly the localization of the centres for the various movements of the lower limb in the spinal cord. These movements are represented in the five lumbar segments and in the first and second sacral segments. According to Mills[126] and Starr[127] the various groups of muscles have their spinal centres as represented in the following table:

First Lumbar Segment.—Iliopsoas; sartorius; muscles of abdomen.

Second Lumbar Segment.—Iliopsoas; sartorius; flexors of knee; quadriceps femoris.

Third Lumbar Segment.—Quadriceps femoris; inner rotators of thigh; abductors of thigh.

Fourth Lumbar Segment.—Abductors of thigh; adductors of thigh; flexors of knee; tibialis anticus; peroneus longus.

Fifth Lumbar Segment.—Outward rotators; flexors of knee; flexors of ankle; peronei; extensors of toes.

First and Second Sacral Segments.—Flexors of ankle; extensors of ankle; intrinsic foot muscles.

These various segments contain the cell bodies of the peripheral motor neurons (multipolar cells of the anterior horns), the axis cylinders of which go to form the anterior nerve roots issuing from these

particular segments. The various nerves, however, it must be understood, do not proceed directly to their muscles but only after having made various connections with the other nerves which go to form the lumbar plexus. Consequently a particular nerve trunk may, and not infrequently does, contain axis cylinders from more than one segment of the spinal cord. It is essential, however, to know the localization of the various motor neurons in the spinal cord on the one hand and to know the motor function of the various nerve trunks on the other. The symptoms of the lesions of these two portions of the lumbar nerves will of course vary, and it is only by comparing these symptoms accurately and minutely that it will be possible to distinguish a focal lesion, involving a few nerve roots, from such a lesion involving a nerve trunk. For instance, a focal lesion involving only the roots of the second and third lumbar nerves would give different symptoms from a lesion involving the trunk of the anterior crural nerve, and this for the very evident reason that, while the anterior crural nerve arises only in part from these segments, and hence might only be partially involved, other nerves, as the external cutaneous, genitocrural, obturator, and even the lumbosacral cord, also receive fibres from these segments and would be partially involved by a lesion of these roots.

The various reflexes have also been located with more or less accuracy in the various segments of the cord. These may be represented in the following table:

FIRST LUMBAR SEGMENT.—Cremasteric reflex.
SECOND LUMBAR SEGMENT.—Cremasteric reflex; knee-jerk.
THIRD LUMBAR SEGMENT.—Cremasteric reflex.
FOURTH LUMBAR SEGMENT.—Gluteal reflex.
FIFTH LUMBAR SEGMENT.—Gluteal reflex.
FIRST AND SECOND SACRAL SEGMENTS.—Plantar reflex.
THIRD TO FIFTH SACRAL SEGMENTS.—Foot reflex; ankle clonus; bladder and anal reflexes.

As in the case of the motor functions so in the case of the reflexes: they are not so strictly localized in individual nerve trunks as they are in the cord segments. Still, by tracing the nerves as carefully as possible through the lumbar plexus to the spinal roots we receive some indication of the reflex function, as it were, of the nerve trunks. Thus the anterior crural nerve is probably the path for the patellar tendon reflex or knee jerk.

In like manner the sensory functions have been localized in the cord. These, according to Starr's table, are as follows:

FIRST LUMBAR SEGMENT.—Skin over the groin and front of scrotum (and upper part of the gluteal region?).
SECOND LUMBAR SEGMENT.—Outer side of thigh.

THIRD LUMBAR SEGMENT.—Front and inner side of thigh.
FOURTH LUMBAR SEGMENT.—Inner side of thigh and leg to ankle; inner side of foot.
FIFTH LUMBAR SEGMENT.—Back of thigh, back of leg, and outer part of foot.
FIRST TO SECOND SACRAL SEGMENTS.—Back of thigh; leg and foot outer side.
THIRD TO FIFTH SACRAL SEGMENTS.—Skin over sacrum; anus; peritoneum and genitals (also over the inner area of the buttocks).

The results embodied in the above table are from observations made especially by Gowers and Ross in England and by Erb and Remak in Germany. Starr, later, collected the numerous facts bearing upon this localization. Ferrier and Yeo in 1881 published the results of their experiments, made by faradizing the several spinal roots in the monkey. These very accurate observations are based upon the best method of proving what movements are represented in the several spinal segments. For sensory tests, however, the lower animals are not well adapted. The pathological data have been furnished by cases of anterior poliomyelitis, syringomyelia, and transverse lesions of the cord, but these for obvious reasons are not always so accurate as those obtained by direct stimulation of the various nerve roots in the monkey. Ferrier's original contribution to this subject appeared in the proceedings of the Royal Society in 1881 and 1883.

In very many lesions of the nerve roots and of the lumbar plexus there are presented to us various forms of combined palsies of the lower limb. This is the case especially in such lesions as traumata of the cauda equina and such as are produced by pressure or septic neuritis following childbirth. In all such cases it will be necessary in order to make an accurate diagnosis to begin, as it were, at the periphery and study carefully the motor, sensory, and reflex phenomena, and then, by grouping these, endeavor to trace them either to the various nerve trunks or to the various nerve roots. It will thus be found not infrequently that the lesions are distributed in such a way as to suggest quite accurately the nerve trunks or even the spinal segments most involved. If the lesion is central, involving either the segments of the lumbar and sacral cord or the nerves of the cauda equina, the symptoms are usually bilateral and have, as it were, a segmental significance. If, however, the lesions are in the nerve trunks, only the muscles and sensory areas innervated by these nerves will be involved. Hence it becomes necessary to take up a careful consideration of the symptomatology of individual nerves.

Diseases of the Iliohypogastric Nerve.

The iliohypogastric nerve is a sensory nerve, distributed by two branches to the skin of the upper gluteal and hypogastric regions. It arises from the first lumbar segment and probably receives fibres from the first dorsal nerve. It could not well be paralyzed alone. Any lesion involving it would probably involve other nerves of the lumbar plexus, especially the ilioinguinal and the connecting loop from the first to the second lumbar nerves. Its paralysis would cause anæsthesia in the upper gluteal and the hypogastric region.

Diseases of the Ilioinguinal Nerve.

This nerve arises in common with the iliohypogastric nerve. It supplies filaments to the internal oblique muscle, and passes out by the external abdominal ring to be distributed to the skin of the scrotum in the male and of the labium in the female and to the upper and inner part of the thigh. The same may be said of it, with reference to disease or injury, as was said of its companion nerve, the iliohypogastric; its paralysis would cause paralysis of the internal oblique muscle and anæsthesia in the territory which it supplies.

Diseases of the External Cutaneous Nerve.

This nerve arises directly from the second lumbar segment and possibly receives filaments from the first lumbar segment by way of the connecting loop between the first and second lumber nerves. It passes into the thigh beneath Poupart's ligament immediately under the anterior superior spine of the ilium. It becomes quite superficial in some places and is distributed to the anterior and outer part of the thigh as far down as the knee, by one branch, and to the outer and posterior surface of the thigh as far as the middle third by another branch.

This nerve might possibly be injured by wounds of the thigh just where it emerges from beneath Poupart's ligament under the anterior superior spine of the ilium. In such a case there would be anæsthesia in the anterior part of the thigh as far as the knee and over the outer and posterior part of the thigh as far as the middle third. Considering its somewhat exposed course it is rather remarkable that more instances are not on record of injury to it. It may possibly be that the result of such injury might be overlooked, as no motor fibres are included in the nerve.

Diseases of the Genitocrural Nerve.

This nerve arises in common with the external cutaneous nerve just described. It passes directly through the substance of the psoas muscle, descending on the surface of this muscle to near Poupart's ligament, where it divides into a genital and a crural branch. The genital branch passes through the internal abdominal ring and supplies the cremaster muscle. In the female it is lost in the round ligament. The crural branch passes along the margin of the psoas muscle, enters the thigh beneath Poupart's ligament, and distributes its sensory filaments to the skin of the upper and anterior aspects of the thigh (Gray).

Because of its close relation with the psoas muscle, which at one place it penetrates, this nerve is probably more frequently involved in psoas abscess than is usually suspected. This is the only lesion in which it would be likely to be injured alone. In such a case there would be abolition of the cremaster reflex with anæsthesia of the upper and anterior aspect of the thigh.

Diseases of the Obturator Nerve.

This important nerve arises by three roots, one respectively from the second, third, and fourth lumbar nerves (Van Gehuchten), or by two roots, from the third and fourth lumbar nerves (Gray). It also penetrates the inner fibres of the psoas muscle; it leaves this muscle at the brim of the pelvis. It then runs along the internal lateral wall of the pelvis to the obturator foramen, where it divides into two branches. It supplies the obturator externus and the various adductor muscles of the thigh. It also gives off articular branches to the hip and knee and sensory branches to the inner side of the thigh and leg, especially by its communication with the long saphenous nerve.

The obturator nerve, as will be seen later, is claimed by some writers (Winckel, Leyden) to be involved sometimes in pelvic exudations resulting from inflammation in puerperal cases. Some authors even claim that it may be damaged by pressure of the child's head during labor, but this is extremely doubtful. In hip-joint disease it is possible that the pain, not infrequently complained of by the patient in the knee, may be due to irritation of the obturator nerve or some of its branches to the hip-joint. The explanation for this pain, however, as given by some writers, as Bonat and Sayre, is that it is due to spasm of some of the muscles of the thigh. It is possible that both these elements, *i.e.*, irritation of the nerve and spasm of the muscles,

may account for this symptom. Injury to the obturator nerve might possibly be caused by wounds of the inner aspect of the thigh, but because of its protected position such injuries are improbable and must be rare.

The symptoms of injury or disease of the obturator nerve are paralysis of the adductors of the thigh and anæsthesia of the inner aspect of the thigh and leg. This anæsthesia, however, is not constant. Paralysis of the adductors interferes especially with the ability of the patient to cross one leg over the other. Outward rotation of the thigh is also impaired.

Diseases of the Anterior Crural Nerve.

This nerve is the largest nerve of the leg, next to the sciatic, and is the largest branch of the lumbar plexus. It arises mainly from a union of the third and fourth lumbar nerves, but possibly receives fibres also from the second lumbar nerve by way of the connecting loop between this and the third lumbar. It passes through the fibres of the psoas muscle and emerges from this muscle at its lower outer border. It passes into the thigh beneath Poupart's ligament to the outer side of the femoral artery. At its point of emergence from the pelvis beneath Poupart's ligament it is a broad, flattened nerve trunk. As it passes into the thigh it divides into cutaneous and muscular branches. The anterior crural nerve supplies within the pelvis the iliacus muscle, and on the thigh the pectineus and all the muscles on the front of the thigh except the tensor vaginæ femoris, which is supplied by the superior gluteal nerve (Gray). It also supplies sensory filaments to the front and inner side of the thigh and of the leg and foot, as well as branches to the knee-joint. The largest of its sensory branches is the long saphenous nerve which descends the leg on its inner aspect. It supplies sensory filaments to the skin of the inner side of the leg and inner side of the foot as far as the big toe. From this distribution it is seen that the anterior crural nerve is largely the nerve governing extension of the leg upon the thigh, and that it supplies sensation to a comparatively large area of the skin of the upper and lower limb.

The anterior crural nerve may be irritated or paralyzed by psoas abscess. This, however, is not a very common symptom. At least it is not often reported. When it is considered, however, that this nerve passes through the substance of the psoas muscle, the suspicion arises that it may be affected oftener than reports indicate. Other abscesses deep within the abdomen may irritate this nerve trunk. Lydston[1] has reported a case of acute lumbar abscess which simu-

lated nephritic colic. In this case a number of cords or branches of the lumbar plexus were involved. The attack came on suddenly with severe pain in the right iliolumbar region. Hence the nerves implicated in this particular case were probably the upper nerves of the lumbar plexus, *i.e.*, the iliohypogastric and ilioinguinal nerves. The case illustrates how readily any of these nerve trunks may be involved by deep lumbar, iliac, or psoas abscesses. Mills'[15] refers to a case of psoas abscess in which a diagnosis of crural neuralgia was made and an abscess was not suspected. Pain extended from Poupart's ligament to the inner side of the knee, following the course of the anterior crural nerve and its internal cutaneous branch. In this case pain was relieved by sitting or lying or flexing the thigh on the pelvis with the knee kept bent. There was also partial paralysis of the iliopsoas muscles, causing loss of power to flex the thigh on the abdomen, the patient being compelled to lift the leg with his hand, especially when he attempted to cross one leg over the other. The anterior crural nerve, from its exposed position at the point where it emerges from beneath Poupart's ligament, might be the seat of injury. Such cases, however, must be exceedingly rare, as but little mention of them is made in medical literature. The index catalogue of the Surgeon-General's office does not give a single reference to injury of the anterior crural nerve. This exemption must be due to the fact that the nerve, while really superficial at the point where it enters the thigh, is nevertheless (because of its location in the groin) very well defended from blows, stab wounds, etc. Gunshot wounds of the nerve do not seem to have been observed. The anterior crural nerve, because it passes above the brim of the true pelvis and not within the pelvis proper, and is well protected by the psoas magnus muscle, is not, as a rule, in a position to receive damage during parturition. Some authors state, however, that the nerve may be the seat of injury during labor. Thus A. M. Fullerton, of the Women's Hospital, Philadelphia, relates the case of a dwarf, aged twenty-five years, with contracted pelvis, in whom the first stage of labor was very slow. The child was delivered with instruments. The mother complained on the following night of great pain in the right groin and entire right limb and was unable to move the limb. Pressure on the anterior crural and obturator nerves caused her to cry out with pain. The symptoms lasted ten days and then yielded to treatment (quoted by Mills'[15]). In this case, however, the fact that the patient was a dwarf with a contracted pelvis probably accounts for the pressure and injury to the anterior crural and obturator nerves, which would not probably have happened in case of a normal pelvis. The nervous symptoms in Fullerton's case are not given in detail, but as the patient recovered in ten days the

injury to the nerve was evidently not severe. The anterior crural nerve, it is said, may also be injured in dislocation of the hip. I find no reference, however, to any such injury in the elaborate study of dislocations of this joint by Allis.[1]" It is not very probable, therefore, that injury from this cause is common.

The symptoms of injury of the anterior crural nerve are highly characteristic. The disability of the leg is very great. If the nerve is involved within the abdomen the iliacus muscle, but not the psoas, is paralyzed. This leads to some inability to flex the thigh upon the abdomen. In attempting this movement or in crossing the affected leg upon its fellow the patient lifts the thigh with his hands. Loss of power of the extensors of the leg is complete. These extensors waste and present the reaction of degeneration. The knee-jerk is lost. Anæsthesia is extensive, involving the anterior and internal aspects of the thigh and the inner side of the leg and foot, the latter because of involvement of the internal saphenous nerve. In irritative lesions pain of a neuralgic character may be very intense and may even be referred to the distal or terminal portions of the nerve. This is so especially in some cases of psoas abscess, as in Mills' case already referred to. In such a case the pain may be relieved by relaxing the psoas muscle, by flexing the thigh on the pelvis, as in the position of sitting or lying. Neuralgic pain in the distribution of the anterior crural nerve has also been observed as an early symptom of a growth near the spine (Gowers).

Diseases of the Sacral Plexus.

The sacral plexus is composed of the lumbosacral cord and the anterior branches of the three upper sacral nerves and of part of the anterior branch of the fourth sacral nerve. It is much more simple in its construction than either the brachial or the lumbar plexus. It is triangular in shape, its base being situated against the sacrum, while its apex points towards the sacrosciatic foramen. It rests upon the anterior surface of the pyriform muscle and is protected in front by the pelvic fascia, which separates it from the viscera of the pelvis. For the clinician it is important to recall that the sacral plexus can be reached by digital exploration through the rectum, and that the chief landmarks are the pyriform muscle, the greater and lesser sacrosciatic ligaments, and the tuberosity of the ischium. With reference to the muscle the plexus lies entirely upon it, and with reference to the sacrosciatic ligaments it is important to recall that they convert the two sacrosciatic notches into foramina, the superior or greater of which is partially filled by the pyriform muscle,

and that through this greater foramen the chief branches of the sacral plexus leave the pelvis.

The sacral plexus has four main branches or nerves—superior gluteal, pudic, small sciatic, and great sciatic. In addition to these it sends off small muscular branches which supply the pyriform, obturator internus, and several other muscles about the true pelvis.

The superior gluteal nerve, the first main branch of the sacral plexus, arises from the lumbosacral cord and is the only nerve of importance that arises from this important connecting link between the lumbar and sacral plexuses. It emerges from the pelvis just above the pyriform muscle by way of the great sciatic foramen, and has a superior and inferior branch. The superior branch supplies the gluteus minimus and gluteus medius muscles. The inferior branch also distributes filaments to these two muscles and then supplies the tensor vaginæ femoris.

The pudic nerve arises from a lower part of the sacral plexus and passes from the pelvis also through the great sacrosciatic foramen, but below the pyriform muscle. Having crossed the spine of the ischium, it reënters the pelvis and is distributed by several branches to the dorsum of the penis, the perineum, and the external sphincter of the anus. It will be described more fully later.

The small sciatic nerve arises by two roots from the lower part of the sacral plexus. It supplies but one muscle—the gluteus maximus—and gives sensory filaments to the skin of the perineum and the back part of the thigh and leg.

The great sciatic nerve supplies the skin of the leg, the muscles of the back part of the thigh, and those of the leg and foot. It is the direct continuation of the lower part or apex of the sacral plexus. Like the pudic and the small sciatic nerve it also passes out of the pelvis through the great sacrosciatic foramen below the edge of the pyriform muscle. These three nerves, as they emerge from the pelvis below the pyriform muscle, occupy the following relative positions: The great sciatic lies farthest exteriorly, then comes the small sciatic, and then the pudic. The superior gluteal, on the other hand, as already said, emerges from the pelvis just above the pyriform muscle. Consequently the pyriform muscle constitutes not only a basis of support to the whole sacral plexus, but also, as it were, a guard or defence for these nerve trunks as they issue from the pelvis at either the upper or lower margin of this muscle.

From this anatomical description it is seen that only one nerve—the superior gluteal—arises from the upper part of the plexus, *i.e.*, from the lumbosacral cord. The other nerves spring from the lower part of the plexus and all emerge below the lower edge of the

pyriform muscle in close proximity to each other. The nerve roots, which go to form the sacral plexus, are with those of the lumbar nerves the largest of all the spinal nerves, and the nerve trunks themselves that form the plexus are also of large size. These nerves can be palpated through the rectum but not through the vagina. The landmarks by which they are to be sought are especially the pyriform muscle and the greater and lesser sacrosciatic ligaments. Because of its deep seat within the true pelvis the sacral plexus is often not so thoroughly explored as it should be in cases presenting symptoms of nerve irritation in the pelvis and about the buttocks and posterior part of the thigh. Its accessibility, however, through the rectum renders direct exploration of its nerve trunks much more possible and convenient than in the case of some other deep-seated nerves of the body. These facts should be borne firmly in mind by the practitioner who has cases that suggest an intrapelvic lesion. It cannot be too strongly impressed upon the minds of all practitioners that thorough pelvic exploration is always urgently demanded in every case simulating sciatica and other neuralgic affections involving any of the branches of the sacral plexus.

The causes of lesions of the sacral plexus are much more numerous than is generally supposed, and as the subject is one of great practical clinical importance it will be discussed here in detail.

The sacral plexus, or the anterior branches of the lumbar and sacral nerves which go to form it, may be injured by tumors or other diseases of the womb, ovaries, and rectum; by inflammatory processes and inflammatory exudates, as in cases of pelvic cellulitis, in abscesses of the pelvis or even of the abdominal cavity finding their way into the pelvis; by neuromata, osteomata, and osteosarcomata; by wounds and pressure during labor, by gunshot wounds, and probably, as in the case of some other nerves, by neuritis of indeterminate cause.

New growths of the womb and ovaries, but especially of the ovaries, may cause pressure upon and irritation of the sacral plexus. The symptoms in such cases vary considerably, and I am convinced that their significance is often for a long time overlooked. These symptoms are too often ascribed vaguely to sciatica or neuralgia or rheumatism about the hip. Thus in one case there was apparently some irritation of the superior gluteal nerve causing a sense of discomfort about the buttock which was probably due to a slight spasm of the gluteus maximus and minimus muscles supplied by this nerve. The tumors that especially cause such symptoms are small ovarian cysts, especially in their early stages, when they may be pressed down firmly upon some of these nerve trunks. I once saw a case in

a lady that illustrated these facts. She was a middle-aged unmarried woman who had been treated for some months for sciatica. It was very evident on close inspection of the hip and thigh that the symptoms did not correspond to those of sciatica. The pain radiated from the buttock around towards the hip, evidently following some of the cutaneous branches of the small sciatic nerve. There was possibly also a slight cramp or spasm in the glutei muscles, as the pain was increased on movement. A pelvic lesion being suspected her consent was finally obtained for a vaginal examination, when a small ovarian cyst not much larger than a large orange was discovered making pressure upon the sacral plexus. This explained all her symptoms satisfactorily and an operation was subsequently performed for her relief. Mitchell refers to a case in a woman in which the patient had numbness in the left foot and leg with loss of power to flex the foot. The peroneal muscles were paralyzed and had loss of electrical response. On vaginal examination a large growth was discovered behind and to the left of the womb. It is probable that symptoms of nerve irritation, especially of the sacral plexus, in cases of tumors of the womb and ovaries, would be recorded more frequently by gynecologists if their attention was more directed to this subject. In such cases careful inspection will usually reveal that the symptoms do not correspond with those of sciatica, which is the disease most frequently diagnosed. Other nerve trunks than the sciatic will be found to be implicated, and in some cases in fact (as in the one just related) the sciatic nerve may even not be involved. In some instances, however, when this nerve trunk is the chief or only one involved, the resemblance to an ordinary case of sciatica may be very striking. In cases in which the superior gluteal and smaller sciatic nerves are involved, the pain on pressure and especially on motion is confined more to the buttock, radiating towards the hip.

Tumors and often affections of the rectum may cause symptoms referable to the sacral plexus. Cancer of the rectum has not infrequently caused such symptoms. Skjeldrup, quoted by Mills,[41] records a case in a man aged fifty, in which rectal examination revealed a tumor. A few years ago a woman was admitted to my wards in the Methodist Hospital, suffering with neuralgic pain about the buttock and thigh. A surgeon, after rectal examination, had decided that there was no organic disease, but that the case was simply one of neuralgia. The woman, however, presented decided appearances of the cancerous cachexia and several enlarged glands were discovered by abdominal palpation in the pelvis. A second rectal examination, more carefully made, revealed the presence of a cancer in the neighborhood of the sigmoid flexure. Immense fæcal accumulations in the

rectum have also caused nerve symptoms referable to the sacral plexus.

Abscesses within the pelvis may cause symptoms of irritation of the sacral plexus or of some of its main trunks. Mills[140] has called attention to the fact that psoas abscess may follow such a course as to involve the sacral plexus. This abscess may follow an irregular or multiple course; thus the pus may escape from the sheath of the psoas muscle, and passing down along the sacrum may leave the pelvis by the sacrosciatic notch. Thompson[142] reported a case of psoas abscess which had been mistaken for a long time for one of sciatica. Severe and intermittent pain followed the course of the sciatic nerve of the right leg. It extended as far as the dorsum of the foot and great toe. Anæsthesia in some territories was also observed. Such pelvic abscesses are not always necessarily connected with diseases of the bone. A man was recently referred to me suffering with so-called rheumatism about the hip and thigh. The nerves, especially about the external aspect of the thigh, were involved. Careful inspection revealed some swelling of the thigh and pain with retardation of movement. Eventually the cause was discovered to be a deep-seated abscess in the pelvis which was evacuated above Poupart's ligament. Some of these pelvic abscesses, however, as already said, may evacuate through the great sacrosciatic foramen. In such a case an early exploration by the rectum would probably reveal the cause of the trouble.

Among the most important causes of injuries and diseases of the sacral plexus and of its main branches is childbirth. The significance of this cause has not always been recognized at its true value and the mode of its action is still a subject of some debate. The subject is not altogether new, although it has been too much ignored by recent writers. Charpentier,[143] in an elaborate monograph, has carefully reviewed this subject from the historic standpoint and given the various facts recorded and the theories by which authors have attempted to explain them. This author, however, ascribes too much importance to the theory of reflex paralysis, as advanced by Brown-Séquard, Bernard, and Jaccoud. Churchill[144] many years ago composed a lengthy article on the paralyses that occur during gestation and childbirth. He collected thirty-four cases from various sources. These cases, however, were a heterogeneous collection, many of them being mere coincidences of gestation and labor, the whole number showing a variety of pathological states. Of these four were cases of paraplegia. These cases of paraplegia appear to have been due to pressure or to septic neuritis or meningitis—one woman, for instance, stood in water up to her knees while at work on the eighth

day after labor (Lloyd[115]). Romberg[116] observed that paralysis could accompany certain diseases of the generative organs, and that it could depend upon direct pressure of the womb or of the ovary upon the sacral plexus; but he attached too much importance to the reflex origin of this paralysis, as advocated by Jaccoud.

Veterinarians have not infrequently observed paraplegia as a complication of metritis in the lower animals (Charpentier). Gellé reported cases of acute metritis in cows after calving, and said that in all his cases there existed a paralysis of the posterior limbs. Swell published an account of an autopsy in the case of a cow afflicted with paraplegia after calving. There were found evidences of inflammation of the uterus and the vagina. Ithen, quoted by Charpentier, has made known many cases of metritis in the mare with loss of power in the hind legs. Imbert-Gourbeyre[117] writes that trauma during labor is a rare cause of paralysis. He gives three cases, however, which seem to have been due to pressure by the head on the sacral plexus or sciatic nerve. Ramsbotham also refers to such cases. Imbert-Gourbeyre admits apparently only two varieties of paraplegia following labor, *i.e.*, the traumatic cases and those caused by myelitis. He ignores very properly the so-called reflex paraplegia. Esnault, as early as 1857, wrote of the paralyses symptomatic of metritis and periuterine inflammation. Leroy D'Etiolles recognizes as a cause of paralysis of the lower limbs not only childbirth but also suppression of the menses, claiming even that pressure upon the sacral plexus could be caused by the latter.

Even in recent years the text-books on obstetrics have little to say on the subject of diseases or injuries of the sacral plexus during childbirth. Cazeaux has a brief allusion to such cases, which he attributes to reflex causes. Winckel[118] recognizes more fully the neuralgias and paralyses of the lower limbs. He says that the puerperal neuroses of the lower limbs are located chiefly in the nerve trunks rather than in the nerve centres and owe their development to parturition. Injurious pressure, he says, is caused by a large head in an unfavorable presentation in a small pelvis. He teaches that pressure may be made by the blades of the forceps and that these may produce severe contusion of the sacral plexus on forced closure as well as during extraction. He recognizes fully a still more important cause for these nervous symptoms, *i.e.*, pelvic exudations or small extravasations which may extend to the sheaths of the nerves. He mentions that parametritis may give rise to such neuralgias and that the location of the affection is usually in the external and middle cutaneous, obturator, and sciatic nerves. Winckel says that even such slight pelvic exudations as accompany phlebitis may compress the nerves

and, finally, that injuries of the vagina with subsequent cicatricial contraction may cause traction and pressure on individual nerve trunks of the true pelvis and that hyperæsthesia and motor disturbances may thus be produced. This statement by Winckel is one of the most comprehensive in any text-book on obstetrics.

Hervieux, however, before Winckel, recognized fully that paraplegia could happen secondarily to an inflammatory lesion of the womb. He cites two instances, of one of which the following is a brief abstract:

A woman 19 years old, in good health, was delivered naturally at the Maternity Hospital. On the sixth day abdominal pains, fetid lochia, and fever announced the presence evidently of some puerperal infection. Two days later a dry tongue, rapid pulse, continued pain in the abdomen with meteorism, insomnia, restlessness, and a general typhoid aspect of the face were present. On the thirteenth day some loss of power was observed. At this time there were intense heat of the skin, pulse 128, meteorism, rapid respiration, abundant lochia, and some erythematous eruption. Later, the rapid pulse and high temperature continued with dry tongue, loss of appetite, sweating, and meteorism and pain in the left iliac fossa. On the sixteenth day absolute inability to lift the lower limbs was noted. When the patient was asked to bend the leg upon the thigh she executed the movement by a zigzag motion of the heel with the object of approaching this part to the posterior aspect of the thigh, but she was not able fully to execute the movement. There was no anæsthesia nor any involvement of the upper limb. The sensibility in the left iliac fossa continued. On deep palpation a phlegmonous induration could be felt and this appeared to be in relation with the adnexæ of the uterus on that side. This patient improved slowly under treatment with blisters and frictions with Neapolitan ointment. As the general state improved the patient began to make some motions with her legs. When she first began to get up she required two assistants to enable her to stand. The phlegmonous induration was gradually absorbed, but the patient continued to complain of vague pains in her thighs. Later there was slight relapse and the appearance of inflammatory hardening in the right iliac fossa. This patient slowly recovered. All sensibility in the hypogastric region disappeared and the woman regained the use of her limbs. When she was discharged from the hospital at the end of two months she was in a good way to recover.

This case of Hervieux,[180] put on record more than a quarter of a century ago, is an important contribution to the subject. Both the report and theory of the author are thoroughly scientific. The patient evidently had septic infection with metritis and pelvic cellulitis, and this phlegmonous inflammation pressed upon and possibly even involved the nerve trunks. As, however, the chief symptom was loss of motor power, the only sensory symptoms being neuralgic pains, it

is evident that these nerve trunks were not gravely involved in a destructive inflammatory process. Pressure was probably the most important factor in causing the symptoms.

Recently Mills[146] has written a useful paper on the subject of puerperal paralyses. Hünermann[147] has also written a paper in which he reviews the literature of the subject. These authors give valuable indications for the systematic clinical study of these important cases. In the discussion of Mills' paper at the College of Physicians of Philadelphia, Hirst said that these paralyses are very rare and that this is the reason why the subject is not treated more fully in the text-books. In a large experience in contracted pelves, difficult labors, forceps deliveries, and head impaction, he had seen but one instance of paralysis of the limbs as a result. He does not subscribe to the view that the paralysis is caused by direct pressure. In a special dissection of the pelvis made to ascertain the reason why nerve injuries are not more common in difficult labor he was impressed with the fact that the nerves are protected by their situation from such injury. The pelvis has a cordiform entrance, according to Hirst, and the nerves are hidden within the bay so that no mechanical pressure is possible. He believes, however, that such an accident might happen in rare cases in which the pelvis has an elliptical shape, but he thinks that nerve irritation and inflammation can occur from a phlegmasia. He has had seven such cases. In such instances the plastic exudate presses upon the nerve and the inflammation extends directly to the neurilemma. Sinkler in the same discussion said that he had come to the conclusion that the majority of cases of neuritis following labor were the result of septic poisoning.

It is possible, however, that pressure may occur upon the sacral plexus in instrumental delivery. Dercum, for instance, has given details of a case of trophic paralysis in a woman after instrumental delivery. There were pain and weakness in the right leg. A second labor, also instrumental and very difficult, caused complete palsy of the right leg, except in the anterior thigh muscles. Two years later wasting with contracture of the affected muscles was present. Hervieux in his work on puerperal diseases refers to paraplegia caused by puerperal poisoning. He thinks that the morbid process may concentrate itself about the sacral region where it is easy to conceive that it might involve the nerve trunks and their envelopes. Campbell in his work on midwifery declares that he never observed a puerperal paralysis except only in one limb, and that this accident always has for its cause the prolonged sojourn of the head in the pelvis, due to disproportion between the capacity of the pelvis and the volume of the fœtal head, and that the lesions are in the pyriformis muscle and

the sacral plexus. Scanzoni believed that in such cases the lesion is the result of pressure, but that the pressure must not be regarded as the only cause, but that there is a more profound derangement. By this he probably means septic inflammation.

Under the head of partial paralyses Charpentier discusses the traumatic origin of some of these puerperal palsies. From a consideration of the facts and authorities which he cites it is difficult not to conclude that trauma, as, for instance, by pressure by the child's head and more especially by the obstetric forceps, does sometimes occur in these cases. Hence a too dogmatic assertion that all these cases of puerperal paraplegia and pelvic neuritis are the results of septic infection should be avoided. It is true that many of the cases reported were studied a long while since, without sufficient critical examination either of their cause or their pathology. But even the older observers were somewhat sceptical of pressure being an active factor in these cases. Accoucheurs, Charpentier says, attribute this accident to a compression of the sciatic (sacral?) plexus and of the obturator nerve, but this action, he says, has not been demonstrated. He thinks it more reasonable to suppose that the accident occurs within the spinal canal, following obstruction to the circulation, etc. But, unable to harmonize this theory with the anatomical facts, he falls back upon the theory of either albuminuria or reflex action.

The following case from Romberg shows clearly the action of trauma.

A woman 33 years old was delivered with the forceps after a difficult labor, which lasted twelve hours. During labor she suffered painful spasms in the left leg, and the following day upon being lifted up she complained of weakness, of difficulty in her gait, and of a loss of sensibility in the left foot. She could not feel the hand when it was passed over the dorsum of the foot nor distinguish the sole upon which it was placed. Motor power was diminished in the foot and limb, which the patient dragged in walking. Cure was obtained in two months.

In this case, which unfortunately is not reported with the accuracy of our more modern clinics, there seems to be a direct connection between a prolonged, painful, and instrumental delivery and the loss of power and sensation in certain nerve trunks. It is noteworthy that these symptoms began even during labor, before the application of the forceps. Thus the woman suffered painful cramps in the left leg, which was the leg eventually paralyzed.

Redemaicher, quoted by Imbert-Gourbeyre, reported in 1852 the case of a woman who was seized with painful but incomplete para-

plegia following a difficult labor, which had to be artificially induced. Cure in this case resulted in eight days, so the inference is that the lesion was slight.

Jaccoud insists that paraplegia may result from compression of the nerve trunks and that this occurs not only in complicated labor, as Hoffmann claimed, but also after regular and natural labor, as Bruns has indicated. Axenfeld said that during labor the head of the child may cause pains in the loins, the thighs, etc., by compressing the nerves distributed to these parts, and that an irritation of the motor nerves, causing painful cramps, sometimes paraplegias, either temporary or persistent, may result (Charpentier).

Bianchi [8] wrote a special treatise, the object of which was to establish the thesis that trauma acts as a cause of paralysis of the lower limbs in women during childbirth. Bianchi regards these traumatic paraplegias as incontestable. He called attention to the fact that similar symptoms can be excited by the pressure of tumors upon nerves, and noted also the analogy of these paralyses with those which Esnault, already referred to, had observed in a case of metritis and pelvic cellulitis. He likened the child's head to a hard voluminous tumor the action of which, although limited in duration, could nevertheless cause decided effects which might persist after labor. Bianchi based his theory upon a consideration of the anatomy of the pelvis, and concluded that it was possible for the head to make pressure upon the nerve trunks. The parts especially exposed to pressure are the muscles of the perineum, the hypogastric vessels, the bladder, the rectum, the nerves, and in particular the lumbosacral cord, the crural, the obturator, and the sacral plexus, especially its great terminal branch, the sciatic nerve. Bianchi contends that the grand sciatic nerve is incompletely protected from injury during labor; that it is forcibly compressed in all labors, but to a variable extent. In ordinary cases this pressure is exerted only towards the termination of the labor, and is shown by cramps in the calves of the legs, but that in some cases long-continued pressure or contusion by the forceps may cause grave and enduring symptoms, and that even true paralyses can supervene in the parts supplied by the sciatic nerve. Bianchi properly calls attention here to the fact that the sciatic nerve is especially liable to be implicated in these cases of prolonged and instrumental delivery. His explanation of the mechanism of labor, by which in the vast majority of cases injury to the sacral nerve trunks is avoided, is clear and convincing. He says that the sacrovertebral angle contributes to lessen the pressure which would be exercised in the sacral excavation, and especially the inclination of the planes and axes of the pelvis directs the principal effort and the great-

est pressure of the head against the anterior wall. Bianchi believes that the special conditions causing traumatic paralysis in labor are the use of the forceps, a posterior position of the vertex, prolonged labor, and finally a contracted pelvis. He cites the following case:

A woman, aged 34 years, had a persistent paralysis of the left leg and foot, following a prolonged and difficult labor. When the head engaged in the pelvis rotation was delayed. The physician made several unsuccessful efforts to deliver with instruments. At every attempt at traction the patient felt a severe pain, fulgurant in character, along the whole extent of the left leg from the hip to the foot. Delivery was eventually effected by perforating the skull. This patient during her lying-in had a double phlegmasia alba dolens. This evidence of septic infection must of course be considered, but Bianchi does not attach sufficient importance to it. The right leg in this case was not affected. In the left leg, the movements of extension and flexion of the thigh upon the pelvis were executed easily. The foot was in a position of extreme extension, its direction being almost in line with that of the leg. The electrocontractility of the muscles, the peroneal and sural group, was abolished, and the sensibility was diminished in the same region. The diagnosis was paralysis of the left leg and foot, caused by a trauma of the sciatic nerve during labor. (Cited by Charpentier.)

From the facts and authorities already cited it is evident that there is some latitude for differences of opinion as to the exact causation of lesions of the sacral plexus and its chief trunks during labor. The older writers were evidently disposed to regard pressure by the head and injuries by the forceps in prolonged and difficult labor as important factors in causing these paralyses. The tendency of more modern writers is to dissent from this view, and to ascribe lesions of the sacral plexus and its branches to a septic inflammation, propagated directly to the nerve trunks from a metritis or a periuterine cellulitis. I do not see that it is necessary to ignore either one or other of these important factors, although I believe that the theory of septic infection is the one that more satisfactorily explains the majority of these cases. There can be little doubt, however, that in case of a large head or a contracted pelvis, the instruments, especially if applied in a faulty manner, might make pressure upon the sacral plexus where it lies upon the body of the pyriform muscle, or especially upon the trunk of the sciatic nerve where it emerges below the lower margin of that muscle and where by reason of its great size and its exposed position it is liable to injury.

Among other causes of injury to the sacral plexus may be mentioned neuromata and sarcomata, and possibly osteosarcomata.

Neuromata of the sacral plexus, while theoretically possible, are practically little if at all heard of. I do not know and have not read

of an instance. Such tumors are not unheard of, although they are rare, in the case of the brachial plexus. An instance of the kind is cited from Duhring in the description of the diseases of the brachial plexus.

Sarcomata and osteosarcomata may occur in the pelvis and may cause symptoms of pressure upon or irritation of the nerve trunks. In such cases, as in all cases of tumors involving nerves, the initial symptoms are likely to be fugacious and deceptive. Thus pain of an intermittent or paroxysmal character may be observed in one nerve trunk. As the case advances this pain becomes more established and may extend to wider areas. It may be associated with other disorders of sensation, such as anæsthesia and paræsthesia. Putnam[18] has put on record a case of sarcoma within the pelvis which involved a number of the nerve trunks. A somewhat rare form of tumor of the sacrococcygeal region comprises the cystic tumors of embryonal or developmental origin which are allied to spina bifida. These congenital cysts may or may not involve nerve trunks. In very many instances these trunks escape. Patients in fact may live to adult life sometimes in ignorance that they are malformed. Emmet and Thomas have both described cases in women in which these tumors gave no trouble before parturition. The nature of these tumors is sometimes quite obscure (Lloyd[19]). They have given rise to much confusion, and a voluminous literature (especially in France and Germany) has sprung up about them. In the article on "Tumors of the Sacrococcygeal Region" in the index catalogue of the Surgeon-General's office, several columns of references are given to papers and monographs on this interesting subject. There are several varieties of these congenital sacral and coccygeal tumors. The coccyx may even be wanting. In some of these cases the morphology is similar to that of spina bifida, the lesion depending upon defective development of the lamina of one or more bones of the sacrum. The sac in these cases, unlike ordinary spina bifida, protrudes anteriorly, hence is located within the pelvis. This may be due to the fact that for some reason this is the direction of least resistance. In some cases, as pointed out by Giraldes, the tumor is located in the perineal region. An important point for diagnosis is the fact that the tumor pushes the rectum before it. In one case reported by Giraldes such a tumor in a woman was punctured by a surgeon and the patient died in consequence with tetanoid symptoms of meningitis. An autopsy showed that the cyst was attached to the sacrum and communicated with the vertebral canal. Emmet and Thomas have each punctured such a pelvic cyst by mistake and both lost their patients in consequence. In such cases the nerves likely to be involved are those

forming the sacral plexus or the branches from it. It is remarkable, however, that in many of these cases the nerve trunks are not involved, and this is due no doubt to the fact that the congenital defect is located at a point below where the main trunks forming the sacral plexus emerges from the spinal canal. Moreover, the tumor being located in the median line does not make pressure upon the plexus itself.

Gunshot wounds of the sacral plexus are rare. According to Mills one hundred and three cases of gunshot wounds of the pelvis occurred in the American war.[163] In one of these the sacral plexus was wounded. This patient, aged twenty-three, was wounded at the battle of Gettysburg. The ball entered the left side of the sacrum and injured the sacral plexus. The leg of that side was paralyzed, but recovery ensued. Details of the case are not given.

Ordinary neuritis of the sacral plexus, due to rheumatism, cold, etc., is probably unknown. Infectious neuritis, such as that occurring after childbirth, undoubtedly exists and has already been described. Mills inclines to the belief that simple non-infectious neuritis of the sacral plexus might occur, but he gives no instance of it in his paper. Considering the protected position of the sacral plexus within the pelvis, it seems to me very improbable that simple non-infectious neuritis of this plexus or its branches should occur. This, of course, does not apply to the main branch of the sacral plexus, *i.e.*, the great sciatic nerve. This nerve, as is well known, is frequently the seat of an acute inflammation, causing the symptoms of the well-known disease, sciatica, which will be described later in detail.

Gray[164] has described two cases of apparent neuralgia of the sacral plexus. The symptoms were pain about the buttock, perineum, scrotum (in a man) and labia (in a woman) and down the back of the thighs, without motor impairment. There was also some anæsthesia. The cause for this condition was not satisfactorily determined, and as no rectal examination was made it cannot be stated positively that there was no organic lesion in either case. As improvement occurred, however, it is not likely that the lesion was grave, and it may simply have been a neuritis due to some obscure causes.

The *symptoms* of disease or injury of the sacral plexus or of its main branches are usually easily determined, although not a few cases occur in which these symptoms are obscure, especially in the early stages of the disease. The obscurity, however, which involves these cases is often more apparent than real, and arises from the fact that the practitioner fails to make a thorough exploration of the pelvis and of the nerves issuing from the great sciatic foramen.

Cases which I have seen and in which the diagnosis had not been satisfactorily made have impressed me with this fact. It cannot be too strongly impressed upon the practitioner that in all cases of obscure pain about the buttock, thigh, and hip, especially in women, an examination of the pelvis is of the first importance. Cases with slight pains about the buttock are too often hastily diagnosticated as sciatica or a simple rheumatism or neuralgia. The patient is not stripped and thoroughly examined. Instances from my own experience have been previously related. Thus, in these cases, in one instance an ovarian tumor, in another cancer of the rectum, and in still another abscess of the pelvis, had all been erroneously diagnosticated as sciatica or rheumatic neuralgia of the thigh. In this connection too much importance cannot be attached to an examination by the rectum. A vaginal examination is often not sufficient to determine the condition of all the pelvic contents. Examination by the rectum allows the observer to make direct palpation of the sacral plexus and also to investigate regions of the posterior and lower regions of the pelvis which cannot be satisfactorily reached through the vagina.

Another important point in the diagnosis of lesions of the sacral plexus is the fact that in most instances they are unilateral, and that even in cases in which they are bilateral they have usually begun on one side and very frequently persist in a worse degree on one side than on the other. This is true even with some of the puerperal cases. Thus in one case, cited above, in which there was a large phlegmonous induration in the pelvis, the symptoms began on one side although eventually the other side became involved.

Affections of the sacral plexus usually present certain types. These have been arranged systematically by Mills[146] and are given here with some slight modification in accord with my own clinical experience: 1. Peroneal type; 2. The sacrodistal and sacrogluteal type; 3. Neuritis, local or multiple, due to septic or other infection; 4. Neuritis with paralysis and pseudo-paralysis due to phlebitis (phlegmasia alba dolens), often septic but having special features.

The first, or peroneal type, of paralysis due to injury or disease of the sacral plexus is one of special interest and importance. Its significance was first pointed out by Hünermann,[147] whose paper is largely devoted to establishing this as a distinct type of puerperal paralysis. The symptoms are about as follows: Usually after protracted and instrumental labor the patient suffers severe pain in the course of the sciatic nerve, with a feeling of numbness and of tingling and creeping on the outer side of the calf, extending to the dorsum of the foot, even so far as the toes. With this is associated a more or

less complete paralysis of the peroneal nerve. The foot cannot be extended (dorsiflexed). Extension of the toes also is impaired and the inner border of the foot is immobile. Flexion of the toes is usually not involved and the movements of the knee and hip joint are not impaired. In some of these cases there is no tactile anæsthesia. This was so in all four of Hünermann's cases, although pain was a well-marked symptom. This pain is described as cramp-like, and in some cases, in fact, cramps are experienced in the affected muscle. In long-standing cases the peroneal muscles waste and the reactions of degeneration occur. The following case from Hünermann is an example of this peroneal type of puerperal paralysis.

A primipara, aged 36 years, was delivered with great difficulty after being in labor three and a half days. Perforation of the head had been required. On the day after her confinement complete paralysis of the right peroneal nerve was observed. The patient had severe pain along the sciatic nerve with numbness on the outer side of the left calf and tingling and creeping sensations in the toes. The paralysis of the peroneal nerve abolished the power of dorsiflexion of the foot and extension of the toes. The movements of the hip and knee joint were not impaired. There was no abolition of sensation. During the first three or four days and nights pain was the most urgent symptom, completely banishing sleep. On the sixteenth day electroexcitability of the paralyzed muscles was clearly demonstrated. The paralysis still persisted when the patient left the hospital at the end of the third week.

Hünermann's three other cases presented symptoms similar to the above. In one patient there was a contracted pelvis, requiring a forceps delivery under ether. In another the patient was also delivered with forceps after a labor of twelve hours, while in the last case (also a forceps delivery) the patient had tearing pains and jerking of the left leg during the use of the instruments. In all these cases pain and cramp were prominent symptoms, with complete paralysis of the muscles supplied by the peroneal nerve. Hünermann's explanation, which has also been adopted by Mills, of the isolated paralysis of the peroneal muscles after instrumental delivery is about as follows: The sacral plexus, as will be recalled, is formed from the first three sacral nerves and from a large nerve trunk coming from the last lumbar nerve, called the lumbosacral cord. The plexus itself lies upon the anterior belly of the pyriform muscle; the first sacral nerve lying at one border (the upper or outer) of the muscle, the third sacral nerve at the lower or inner border, the second sacral nerve lying between them. The plexus thus rests on soft muscular tissue. The muscle itself passes freely through the large sciatic foramen of the pelvis, where it is not subject to pressure against a bony surface in

such a way as to involve any of the nerve components of the plexus. In the case of the lumbosacral cord, however, which enters into the formation of the sacral plexus, the case is different. This cord arises largely from the fifth and partly from the fourth lumbar roots. It is a very large cord and as it passes down into the true pelvis it bends over the sharp border of the true pelvis. This lumbosacral cord is therefore more liable to pressure and injury than the other parts of the sacral plexus, especially in a high-forceps operation. Now this lumbosacral nerve, according to Lefebre and others, is the root of the peroneal or external popliteal nerve. This nerve in fact is a continuation of the lumbosacral cord, and in rare instances it has been traced as a separate nerve all the way from the pelvis (Mills). As will be recalled, the superior gluteal nerve arises from the lower part of the lumbosacral cord. This nerve supplies the gluteus medius and minimus muscles. From its origin, it is liable in these cases to injury, and this has been found to be the case in some instances. The distribution of the peroneal nerve is as follows: It divides into the anterior tibial and musculocutaneous nerves. The anterior tibial supplies the tibialis anticus, extensor longus digitorum, extensor proprius hallicis, and extensor brevis digitorum. The musculocutaneous nerve supplies the muscles on the fibular side of the leg, and gives branches to the peroneus longus and the peroneus brevis muscles. The sensory filaments of the peroneal nerve by its two main branches supply the great and second toes and the adjoining sides of the second and third toes, the dorsum and inner side of the foot, and the inner side of the ankle; also the outer side of the foot and ankle and all the toes excepting the outer side of the little toe. As a consequence of this distribution the paralysis in this peroneal type of puerperal palsy involves the muscles which dorsiflex the foot and extend the toes. The sensory area involved includes most of the toes, the dorsum of the foot, the outer side of the ankle, but, as already said, anæsthesia is usually not marked in these cases. It is found from clinical experience that the tibialis anticus muscle is most likely to remain paralyzed. This muscle elevates the inner border of the foot, extending (dorsiflexing) and adducting the foot at the ankle. The extensor longus digitorum extends the toes and dorsiflexes the foot. The peroneus longus and peroneus brevis muscles evert the foot and rotate it outwards. By reference now to the table given elsewhere of the representation of the various muscles in the different segments of the cord (see page 249) it will be seen that those muscles affected in puerperal traumatism have their centres in the fourth and fifth lumbar segments, and it is from these segments that the great lumbosacral cord arises. Hence the demonstration

here given, according to Hünermann and Mills, is clear, that in those cases of paralysis following the high-forceps operation this lumbosacral cord is the portion of the sacral plexus most frequently involved, and that this is by direct pressure or injury. A further proof that this paralysis is caused by injury, and not by infectious neuritis extending from a metritis or pelvic cellulitis, is the fact that in some of these cases the paralysis appears even as early as the first day after delivery. An additional proof of the mechanism of this paralysis is the fact that the lumbosacral cord is not located in a favorable position to be attacked alone by an infectious process arising from a septic inflammation about the uterus. Any such septic process causing pelvic cellulitis would be quite as likely, if not more likely, to involve the sacral plexus proper.

It is supposed by some authors that this involvement of the lumbosacral cord may cause an ascending neuritis, which eventually involves the spinal cord. This hypothesis, however, is not necessary to explain the clinical fact. The paralysis is so distinctly localized in the distribution of the peroneal nerve that it is satisfactorily explained by involvement of this large nerve trunk alone. In cases in which the paralysis passes from one side to the other, it is likely that an inflammatory process has been set up in the cellular tissue surrounding the lumbosacral cord, and that this passes in time to the other side. This seems to me a more reasonable explanation than that given by Mills, that the inflammation ascends to the roots of the cauda equina and passes from one to the other by reason of their contiguity. If this were so other nerve trunks would also be involved. It is also possible in these cases, in which the affection seemingly passes from one side to the other, that the injury has originally been bilateral, but because of the position of the child's head the injury is worse and therefore more promptly manifested on one side than on the other. It may be recalled, however, by reference to Hünermann's cases, that the symptoms were almost uniformly unilateral. The one striking exception to this occurred in a woman in whom there was a suspicion of syphilis.

In discussing the mechanism of this peroneal type of puerperal palsy Hünermann does not incline to the belief altogether that the trauma to the lumbosacral cord is caused directly by the forceps. These cases, it is true, usually happen after forceps delivery, but they occur in women who, from contracted pelvis or some other cause, have prolonged and difficult labor. This renders an instrumental delivery necessary, but the immediate cause of the compression or bruising of the lumbosacral nerve is pressure by the child's head. Hünermann believes that rotating or side-to-side movements

effected by the forceps are especially liable to cause this accident. Mills inclines to the belief that a practical point in the differential diagnosis of nerve injuries by the forceps from those caused by pressure of the head of the child is that in the former the sacral nerves are most likely to be involved, and in the latter the lumbosacral cord and possibly the first sacral nerve. In Hünermann's cases certain important obstetrical data were obtained with reference to the position of the head and the size and shape of the pelvis. The measurements, according to him, showed a generally contracted pelvis. He believes that these accidents are not liable to occur in a normal pelvis except in face and brow presentations. It is a remarkable fact that in Mills' case two sisters of the patient died in labor within two months of her own delivery. This seems to point to the possibility of all three sisters having deformed pelves.

A proper scientific study of this injury no doubt demands more careful investigation of the diameters of the pelvis. It is evident that some forms of contracted pelves are more liable to lead to crushing of the lumbosacral cord than others. Hünermann, for instance, states that the flat rachitic pelvis is not liable to cause this accident since the oblique diameters of this pelvis are greater than the average.

In the second or sacrodistal and sacrogluteal type of puerperal palsy the symptoms are merely of a neuralgic character in certain well-defined areas, without paralysis or with very slight paralysis of short duration, or even with only a pseudo-paralysis due to the inhibition of movement by pain. These cases probably do not differ essentially from the preceding, except in the severity of the symptoms and the predominance of pain. The symptoms are not always confined, however, to the distribution of the lumbosacral nerves. They may be called in fact preëminently the neuralgic type of puerperal palsy. In these cases there is usually considerable pain during labor. This persists after labor and may even be aggravated for a day or two. The character of the pain is severe and it is usually associated not so much with true paralysis as with immobility of the limbs caused by the pain. The location of this pain varies. It may be felt in the groin and may even seem to involve the entire limb. In these cases the anterior crural and obturator nerves may be involved, as well as the branches of the sacral plexus. Thus in Fullerton's case, already referred to, pressure on the anterior crural and obturator nerves caused the patient to cry out with pain. If the nerves of the sacral plexus are involved the pain may radiate about the buttock and down the posterior aspect of the thigh. In such cases exploration by the rectum may lead to the detection of the individual nerve trunk or trunks of the sacral plexus

that are involved. Pressure upon them will usually cause the patient to cry out with pain. In cases of this type there is not usually any true anæsthesia. Mills records a case of this sacral neuritis in a woman aged thirty-three. When first pregnant she began to suffer pain, extending from the hip to the heel of the left side. After labor she did not entirely recover from this, and with each succeeding pregnancy the pain became worse. After the birth of the fifth child she was obliged to use crutches because of the pain in walking. There was ultimately some slight sensitiveness also in the other leg. This sensitiveness in the leg first affected was found particularly over the sciatic distribution. Examination by the rectum and the vagina showed extreme sensitiveness of the left sacral plexus. There was also found a prolapsed, enlarged, and retroverted womb, with pelvic exudations and adhesions. The ovarian tube on one side was tender and enlarged. In this case there was evident a general inflammatory state, or the results of an inflammatory state, in the pelvic organs. This had resulted evidently in involvement of some of the nerve trunks, with resulting neuralgic symptoms.

Kelly, quoted by Mills, refers to a case of a woman who had suffered with exacerbations of great pain in the pelvis, which had led eventually to the removal of both tubes and ovaries. This had not relieved her pain. Careful examination subsequently showed that the uterus and its surroundings were free from disease. By rectal examination, however, it was found that one of the main cords of the sacral plexus was exquisitely sensitive, so that when it was touched the patient gave a sudden scream, at the same time doubling up her leg. The disease of the sacral plexus in this case had followed a difficult forceps delivery.

All cases of this neuralgic type of disease of the pelvic nerves are not due necessarily to labor and its complications. Disease of the ovaries and tubes may also cause such symptoms. Ovarian tumors and diseases of the rectum, as already explained, may cause obscure neuralgic pains in the nerves issuing from the pelvis.

The third type—neuritis, local or multiple, due to septic or other infection—is referred to here briefly as throwing some light upon the genesis and pathology of these puerperal palsies. It does not in strictness, however, belong to a consideration of disease of the sacral nerves, although these nerves are usually involved. In this class of cases there is usually a more or less diffused or multiple neuritis, the nerves involved not being necessarily only those of the lumbar and sacral plexus. In other words there is an inflammation of nerve trunks due to the absorption into the blood of septic matters from the womb. That various poisons may cause a multiple neuritis is now

well known. I reported a case with Dr. Riesman[166] in which a general multiple neuritis was evidently caused by a septic condition of the blood, associated with an endocarditis, although the case was not a puerperal one. Some writers have reported cases of multiple neuritis occurring after normal labors. In some of these cases it is more than probable that the cause of the disease was alcohol. The earlier symptoms may even occur before labor, and in such cases a true septic origin cannot well be supposed. Cases that occur after labor usually come on before the end of the third week. In cases in which there is a true septic origin the nerves of the pelvis are usually involved early. Later other nerves in other portions of the body may become involved. It is proper to say, however, that such instances are very rare up to date in medical literature, and cases that have occurred or that may yet occur require or will require a very searching criticism to establish their identity. While it cannot be denied that, theoretically, puerperal infection may cause a more or less diffused multiple neuritis in other portions of the body, still the alleged fact must be accepted with caution. Direct involvement of the pelvic nerves in an infectious cellulitis is readily understood, and so may be an inflammation of other and remote nerve trunks by septic poisoning carried to them from the blood. Practically, however, we know that while the former instances are not uncommon the latter are extremely rare. The necessity of a careful criticism in these cases is well shown in the case reported by Möbius[168] in a woman who was seized with painful cramps in the left calf three weeks after the birth of her child. Loss of power in the forearm and in the scapular region occurred later. In this case, as quoted by Mills, alcohol and lead are not excluded as possible factors. Handford's[167] cases show unmistakably the action of alcohol. There was nothing special about them except the fact that the disease came on during the puerperium. Both women were wives of publicans and had evidently been addicted to the excessive use of alcohol.

The fourth type is a form of neuritis, with paralysis and pseudoparalysis, due to phlebitis, often septic but having special features. In cases of pelvic phlebitis or phlegmasia alba dolens the patient may have involvement of some nerve trunks and may be for a time partially paralyzed. Neuritis may result from spread of the inflammation by contiguity or by pressure. In cases in which neuritis occurs the symptoms of nerve injury may possibly persist after the other symptoms have subsided. These cases, in fact, are instances simply of an infectious process, and hence do not differ materially from cases already mentioned except in the fact that the principal veins are involved. Thus a patient suffering with phlebitis may develop other

septic symptoms. She may become pyæmic, and an infectious myelitis, resulting in an incurable paraplegia, may result. Winckel quotes authorities to prove that in some cases of phlegmasia paralysis of the affected leg may remain. In such cases there is usually a history of some septic condition occurring during or subsequent to labor. In one case, referred to by Fullerton, an abscess had formed over the right sacrosciatic foramen. A left crural phlebitis existed, with the characteristic œdematous and anæsthetic condition of the leg. The uterus was embedded in a mass of pelvic exudate. In such a case the impairment of movement is largely due to the swollen state of the leg, and is not a true paralysis. As, however, there was extensive septic infection of the pelvic organs it is possible that some of the pelvic nerves may have been involved. In some cases localized or diffused pain may exist in the leg. Thus the great toe or the foot may be the seat of neuralgic pains. In some cases the limb may not only be paralyzed but wasted with some loss of sensation. This condition is probably due to injury or inflammation of some of the nerve trunks in the pelvis, which is only coincident with the phlegmasia, that is to say, the conditions are all dependent on a septic infection. From examination of reported cases, especially those quoted by Mills, it is apparent in this type of case, in which phlebitis occurs, that a true paralysis, due to injury of the nerve trunk, is not always present, but that the disability of the leg is due to the swelling and consequent stiffness.

The *diagnosis* of all forms of disease or injury to the sacral plexus depends of course upon a thorough exploration of the parts. As already said, a thorough exploration of the pelvis by the vagina and rectum is often essential in obscure cases to clear up the diagnosis. Examination by the rectum is especially important. By this means the nerve trunks can be palpated and any disease, as tumor or inflammation, in the posterior part of the pelvis can usually be demonstrated. It may be put down as a rule, that in every case simulating sciatica or neuralgia about the hip, groin, buttock, or thigh an examination of the pelvic contents should be made.

The distribution of the pain and paralysis in the different types of disease just described is usually sufficiently characteristic. These symptoms can generally be traced to involvement of one or other of the main nerve trunks of the sacral plexus. The history of the case, especially of a preceding labor, with instrumental delivery or septic complication, often clearly indicates the character of the lesion. In cases of beginning ovarian tumor or rectal disease, however, mistakes are easily made, as the preceding history of the case may be obscure. In such cases exploration of the pelvis is imperative.

Hysteria can unquestionably simulate organic disease of the pelvic nerves. Churchhill, for instance, in his paper already cited, gives undoubted examples of hysteria occurring in the puerperium. Leeson reports two cases of paralysis after adherent placenta, which were evidently hysterical; contractures were the prominent symptoms, and the women recovered promptly. In such cases characteristic hysterical stigmata are usually present, especially segmental anæsthesia of the leg or hemianæsthesia involving both arm and leg. Characteristic mental states are also present. The affected muscles do not degenerate, and the paralysis is not confined to the muscles supplied by any one particular nerve, but usually involves all the muscles of the leg.

The *pathology* of these affections of the sacral nerves is readily understood and has been indicated sufficiently in the special section devoted to the subject of the general pathology of the nerves. In cases of simple pressure or crush, as those in which the lumbosacral cord is involved, the nerve fibres are undoubtedly ruptured. In cases of septic infection of the pelvic organs and tissues the nerves are no doubt in a state of more or less acute inflammation, or they are embedded in an inflammatory exudate and thus subject to pressure.

The earlier theories, first of a functional, and second of a reflex origin, for these puerperal paralyses are now recognized as no longer tenable. This latter theory, which was long upheld by the older observers, rested upon the belief of an abnormal excitation which exhausted the spinal centres, and thus, as it were, paralyzed the nerve trunks. Such a theory was little better than a cloak for ignorance, and does not require serious consideration here. The theory of an ascending neuritis, passing eventually into the spinal cord, is still held by some observers and is possibly not unreasonable. As the trophic centres for both the motor and sensory neurons are located far above the seat of inflammation, it is not likely that an ascending degeneration can occur. According to Fleming, however, distinct changes do occur in the cell-bodies, both in the spinal ganglia and in the anterior horns, in cases in which the axis-cylinders have been cut across or compressed. Those changes consist in a shrinking of the nucleus and a shifting of its position, and in apparent alterations in the chromatic elements in the cell. In the case of acute inflammation the possibility of this process ascending even to the spinal cord cannot be denied. The symptoms, however, in most of these cases do not indicate clearly an involvement of the spinal cord. The bladder and rectum are usually not involved, and the symptoms are merely such as can readily be accounted for by implication of the nerve trunks.

The *treatment* for all forms of paralyses of the sacral nerves is a subject of great importance. Much, of course, will depend upon the accurate diagnosis of the case and the determination of the cause. If neuralgic symptoms are excited by organic lesions deep within the pelvis, it will be useless for the practitioner to address remedies to the cure of the symptoms instead of to the removal of the cause. Thus sedative, anodyne, and alterative drugs will prove of practically no benefit in a case of ovarian tumor making pressure upon some of the nerves of the pelvis. On the other hand, a surgical operation for the removal of diseased tubes and ovaries, the seats of septic infection, will not necessarily cure a neuritis that is dependent upon a transmission of this septic process through connective tissue to the nerve trunks.

Of first importance, of course, in obstetric cases is thorough asepsis. This is of more importance even as a prophylactic than as a therapeutic measure. The discussion of this subject, however, is more appropriate in special works on obstetrics. Surgical treatment in such cases is often required promptly. The evacuation and drainage of pelvic phlegmons, the ablation of diseased ovarian tubes, the thorough irrigation and cleansing of the whole parturient canal, are among the procedures demanded in such cases. The mischief once done, however—*i.e.*, an inflammatory process once having fastened upon the nerve trunks—the ordinary obstetric procedures and operations will not necessarily cure the patient promptly or even eventually. The neuritis under these circumstances may be as obstinate to treatment as in the case of nerve trunks in other parts of the body, and we know that neuritis, especially when set up by trauma and infectious processes, is usually a stubborn disease. Still the ordinary obstetric procedures and even major operations are in the line of proper treatment for many of these cases.

In case of malignant disease, such as cancer of the rectum or sarcoma of any part of the pelvis, as in Putnam's case, the question of operation is a purely surgical one. The primary disease in these cases usually so greatly overshadows the nerve lesions that the question of treatment of the latter, except by anodyne drugs, seldom arises.

In all forms of puerperal paralysis rest is absolutely essential. These patients should be kept upon their backs in bed; this is usually indicated in all forms of puerperal sepsis, but especially in cases in which the nerves are involved. Absolute immobility of the limb, more particularly in grave cases, such as the peroneal palsy due to injury of the lumbosacral cord, is indicated. Mills even recommends that sand bags be applied to either side of the legs and feet in

order to secure this immobility. Fullerton elevated the limb and bandaged it with flannel. This observer also applied flaxseed poultices over the groin in a case in which the anterior crural and obturator nerves were involved. In some cases, instead of using sand bags, it is more desirable to put the affected limb upon a splint. By this means the leg can be kept more securely fixed than in any other way. It is desirable also to swathe the leg in cotton. This maintains the circulation and promotes sweating.

Alternate hot and cold bathing of the leg is recommended by some. As, however, the morbid process is deep seated within the pelvis it is difficult to give a rational explanation for such treatment to the periphery; nevertheless, experience seems to prove that such bathing is sometimes beneficial. It probably acts in several ways. Thus it may both maintain the circulation and also favorably impress in some way the nutrition of the nerve trunks.

Anodyne and sedative drugs will almost invariably be demanded in these cases because of the pain and the general nervous reaction of the patient. Opium undoubtedly is the most efficient drug for this purpose, but it should be used with caution lest it lead to the formation of the drug habit. This is especially true in the graver forms of these paralyses in which the prospect for cure is remote. The patient under these circumstances will soon come to rely upon an opiate in some shape. In the milder cases, however, as, for instance, those in which simple neuralgic pains are present, a proper use of some opiate is indicated. The drug probably acts not only as a palliative but even as a curative by assisting in keeping the nerve trunks perfectly at rest. Thus in cases in which the natural tendency of the disease is to abate and even disappear in a few weeks, there is no good reason why the patient should not have the benefit of the relief and possibly curative action of opium in some form. The best methods of administering the drug in these cases are by opium suppositories and the hypodermic use of morphine. Among other sedatives, the coal-tar products, such as phenacetin, antipyrin, antikamnia, or exalgin may be employed.

Of alterative drugs, salicylate of sodium or salophen is probably efficacious in some instances. Iodide of potassium is of little use in cases of injury or inflammation of the sacral plexus. Some authorities recommend the use of mercurials in these cases. Inunctions of mercurial ointment may be used on the affected leg, especially upon the inside of the thigh and in the groin. This treatment may even be persevered with until a slight constitutional effect, shown by a mild degree of ptyalism, is secured.

After the acute stage is past an endeavor should be made to

restore the paralyzed muscles. The best remedies for this purpose are massage and electricity. In cases in which the muscles have degenerated and the reactions of degeneration are present, the faradic current is not appropriate. The muscles will not react under these circumstances to this current. Galvanism should therefore be employed in a current of sufficient strength to cause slight muscular response. Massage of the affected limb is undoubtedly a useful adjunct to the galvanic treatment. It should be done by an expert, and only with great care and gentleness, especially in cases in which the muscles have undergone advanced degeneration.

Diseases of the Superior Gluteal Nerve.

This nerve, as already described, arises from the lumbosacral cord. It emerges from the pelvis through the great sacrosciatic foramen, above the pyriformis muscle, being the only nerve of the sacral plexus that passes out above this muscle. It is distributed by its two terminal branches to the gluteus medius and minimus muscles and to the tensor vaginæ femoris. It is entirely motor.

This nerve is seldom paralyzed alone. As it arises from the lumbosacral cord it may be involved in injuries to this cord, such as have already been described as occurring in difficult cases of labor. Thus in all cases of the peroneal type of puerperal paralyses injury to this nerve may be suspected and the symptoms of it should be sought for. Paralysis of the muscles supplied by this nerve causes inability to rotate the thigh inwards. There is also impairment in the ability to gain an erect posture after stooping and in rising from a sitting position.

The treatment of this affection has already been indicated under the head of the sacral plexus.

Diseases of the Pudic Nerve.

This nerve arises from the lower part of the sacral plexus, and like all other branches of this plexus, excepting the superior gluteal nerve, passes out of the great sacrosciatic foramen below the lower edge of the pyriformis muscle. By its various branches it is distributed to the skin about the anus, the perineum, the back of the scrotum, and the penis or clitoris. It gives muscular branches also to the transversus perinei, accelerator urinæ, erector penis, and compressor urethræ. Paralysis of this nerve causes anæsthesia in the parts indicated, and some loss of power in the bladder, which, however, is usually not complete. An important point of distinction is that the side of the scrotum is not anæsthetic in case of paralysis of the pudic

nerve. This is due to the fact that this portion of the scrotum is supplied by the ileoinguinal nerve, which arises from the upper part of the lumbar plexus. The pudic nerve is seldom if ever involved alone. In lesions of the sacral plexus, however, it may be paralyzed or irritated. Characteristic symptoms are neuralgic pains about the anus and perineum.

Diseases of the Small Sciatic Nerve.

The small sciatic nerve is distributed to the skin of the perineum and to a well-defined area on the buttock, the posterior aspect of the thigh, and leg. It supplies only one muscle, the gluteus maximus. It arises by two roots from the lower part of the sacral plexus. It passes out of the pelvis in company with the great sciatic nerve beneath the lower margin of the pyriformis muscle. This nerve supplies a well-defined area or strip of skin over the gluteus maximus, the perineum, the posterior aspect of the thigh, the popliteal region, and the upper and posterior part of the leg.

The small sciatic nerve may be and has been injured alone. In one case, recently under observation, I saw well-defined symptoms of injury of this nerve trunk as a result of an accident caused by the collision of two trolley cars. In this case the patient had been violently shocked in the collision and had been picked up unconscious. There were large ecchymoses under the skin of the loins and flank. After recovery from the immediate effects of the accident the patient continued to suffer for many months with well-marked anæsthesia over the posterior part of the thigh and in the perineum. There was a slight limping gait, such as might be caused by a wrench or strain of some of the muscles of the hip and thigh. The patient also had some loss of virile power. While all these symptoms could not be attributed exclusively to injury to the small sciatic nerve, yet the anæsthesia in the distinct and limited area showed that that nerve at least had been injured, along possibly with some other nerve roots or components of the sacral plexus. In such a case it is impossible to state dogmatically just what the injury to the nerve is or where exactly it is located. In these traumatic cases, however, it will generally be found that single nerve trunks are not involved alone. The injury, whatever it is, usually implicates the fibres of several nerve trunks or nerve roots. As these fibres are variously combined in the sacral plexus, the possibility is that the lesion is located either in that plexus or, more probably, in the roots of the sacral nerves within the spinal canal, or in the sacral nerves themselves as they emerge from the sacral foramina.

The lesion itself in these cases probably consists in minute con-

tusions or even capillary hemorrhages within the nerve trunks. We must suppose that there is distinct organic change in the nerves that produces such persistent symptoms. It does not do to suppose a mere dynamic lesion. The lesion is doubtless organic or structural, although exceedingly minute. Even the rupture of a few axis cylinders is quite conceivable as the effect of profound shock or concussion. Subsequent attempts at repair cause a hyperplasia, which in turn may press upon and obliterate other nerve fibres within the same nerve trunk. A kind of scar tissue may thus be formed by which the nerve is possibly for a long period, or even irreparably impaired. Such a pathological process seems essential to explain some of these traumatic cases. The nerves involved are especially those of the dorsal, lumbar, and sacral regions. Such cases are often of great medico-legal importance. They are the cases in which claims for damages are not infrequently put forward, and are sometimes very justly allowed by courts and juries. As the symptomatology in these cases, however, is often not a little obscure, it is essential that the patients should be examined with the utmost minuteness of detail and with knowledge of the exact course and distribution of the main nerve trunks. The case above referred to was an example of the necessity for such care. By eliciting and locating all the obscure sensory and motor symptoms in these cases, they often gradually assume a clear and distinct type, which can frequently be successfully demonstrated even to the lay mind.

The small sciatic nerve may be involved in the various lesions of the sacral plexus, which have been already described and need not be repeated here.

The *symptoms* of disease or injury of the small sciatic nerve are anæsthesia of the skin covering the posterior part of the thigh, the glutei muscles, the perineum, and the upper and back part of the leg. There is also paralysis of the gluteus maximus muscle, and this is shown especially by inability or impairment of the power of the patient to arise from a sitting position. His ability to stand upright is not impaired.

The *treatment* of disease or injury of the small sciatic nerve has been in the main sufficiently indicated in the section on the sacral plexus. A few words may be said, however, about the medico-legal traumatic cases. Patients suffering with these lesions often require great tact and discrimination in their management. There is nearly always present a subjective or hysterical element in such patients which may seriously complicate the diagnosis and baffle the treatment. Therefore, while fully recognizing the fact that the patients have organic lesions, the practitioner should in every

instance make a proper use of mental or suggestive therapeutics. The subjective element in these cases is no doubt intensified and maintained by a pending lawsuit. Consequently a guarded prognosis should be given, with reference both to the duration of the case and to the ultimate recovery, so long as the lawsuit is unsettled.

Diseases of the Great Sciatic Nerve.

This, the largest nerve in the body, arises from the apex, as it were, formed by the junction of the various cords of the sacral plexus. It passes out of the pelvis through the great sacrosciatic foramen beneath the lower margin of the pyriformis muscle. Occasionally there is an anomalous high division of the great sciatic nerve before it emerges from the pelvic cavity. In such cases one branch or division of the nerve passes directly through the body of the pyriformis muscle, the other division pursuing the usual course beneath the margin of that muscle. In emerging from the pelvis the nerve is located about midway between the tuberosity of the ischium and the trochanter major (rather nearer the former), where it is easily subject to pressure and palpation. It descends beneath the surface of the back part of the thigh, almost in the median line, being covered in the upper part of its course by the gluteus maximus muscle, and, about the middle of the thigh, by the biceps. Except in the anomalous cases just referred to, it divides at the lower third of the thigh into its two main branches, the external and the internal popliteal. In the anomalous cases in which this division occurs within the pelvis the two nerves descend side by side in the thigh. The great sciatic nerve is both sensory and motor. In the thigh before its division it supplies the flexors of the leg, *i.e.*, the biceps, semitendinosus, semimembranosus, and adductor magnus. By the internal popliteal nerve it supplies the sural group of muscles, as well as some of the smaller muscles of the foot. By the external popliteal or peroneal nerve it supplies the tibialis anticus, extensor longus digitorum, extensor proprius pollicis, peroneus longus, and peroneus brevis. The great sciatic nerve supplies sensory filaments to the outer half of the front of the leg, to the outer lower part of the back of the leg, the dorsum and sole of the foot, also to the little toe and the neighboring half of the fourth toe. These distributions will be described more minutely when the diseases of the two main branches of the nerve are discussed.

The main trunk of the sciatic nerve may be injured by pressure and by various wounds. It may also be involved in tumors and may be the seat of special inflammatory or irritative lesions which pro-

duce the symptoms of the well-known disease called sciatica. This disease will be described presently.

Numerous instances are on record of injuries and wounds of the sciatic nerve. Annequin[100] has recorded an instance of section of the trunk of the sciatic nerve at its point of emergence from the pelvis. Bouilly[100] relates a case of contusion of this nerve. Charcot has also put on record a similar case. Park [100] gives an instance of rupture of the sciatic nerve. Van Buren[101] saw a case of wound of the great sciatic nerve, which caused a trophic ulcer in the sole of the foot, and Warren[102] has narrated an instance of gunshot wound of the thigh in which the great sciatic nerve was implicated.

The details of these cases of injury to the sciatic nerve are full of interest and instruction, from both the physiological and the therapeutic standpoints. Banner[103] relates a case of injury to the sciatic nerve in a man, caused by the following accident: The patient fell out of a window, striking upon the iron spikes of a fence surrounding an area below. He received two punctured wounds, one involving the anus and the other on the back of the thigh. From this latter wound protruded filaments of the sciatic nerve. The principal symptoms were pain and anæsthesia in the course and distribution of the sciatic nerve and spasmodic twitching of some of the muscles supplied by this nerve. The whole nerve had evidently not been divided, but it had been lacerated and part of it had been partially stripped off of the main trunk and was protruding from the wound. The spasmodic twitching of the muscles could be explained by the fact that they were still in connection with the nerve, the fibres of which were irritated by the injury. Recovery in this case was almost perfect. Warren[102] reports the case of a man aged eighteen years who, at the battle of Williamsburg, in the American Civil War, received a bullet wound midway between the tuberosity of the ischium and the great trochanter of the left side. He suffered but little pain at first, but in a few days great pain began in the sciatic nerve. The limb was drawn up and held immobile, but this seems to have been partly due to the pain. An exploratory operation revealed spicules of bone pressing against and irritating the great sciatic nerve. The case is imperfectly reported, as details of motor and sensory symptoms are not given; but it is possible that the nerve trunk was not severed but only wounded by the bullet or irritated by the spicules of bone. Park [100] relates a unique case of rupture of the sciatic nerve in a boy aged thirteen years. The patient had had an excision of the hip-joint for severe coxitis. The disease had recurred and progressed. Extensive sinuses had formed and there was tuberculous caries of the ischium. At a second surgical operation a large flap of the buttock was raised, thus exposing the

sciatic nerve, which was found adherent to surrounding tissue. While the surgeon was dissecting the nerve away, the patient suddenly aroused from the anæsthetic and gave a violent wrench, completely rupturing the nerve. The ends were immediately joined and sutured. Complete paralysis of motion and sensation occurred in the sciatic distribution, but perfect recovery followed in two weeks. This claim of entire recovery of motion and sensation in two weeks after complete rupture of the sciatic nerve is difficult to reconcile with what we know of the anatomy of the motor and sensory neurons, and of the slow process of regeneration that must necessarily take place after the axis cylinders have been divided. Like too many cases of injuries to nerves reported by surgeons, the symptoms are not given with sufficient detail to admit of a strict criticism. In Van Buren's case[104] a man aged twenty-two had received a wound from a small sword which passed entirely through the right thigh at its lower third behind the femur. Loss of sensation in the skin on the peroneal side of the leg and foot, together with loss of power of motion of the foot and of the toes, followed the injury. These symptoms continued at the expiration of two years when the patient was observed. In the second year after the accident a trophic ulcer appeared on the heel. This began as a blister, which soon degenerated into an indolent ulcer, which had lasted for a year. It was about two inches in diameter, extending in a conical form down to the os calcis. It healed under appropriate treatment, continued for two months. Annequin's case[105] was briefly as follows: A man aged twenty-one years received a wound from a stiletto in the posterior part of the left thigh. Severe pain followed, and eventually there was wasting of the left leg, paralysis of the foot, and loss of the power of flexion of the leg upon the thigh. There was also impairment of sensation. There was paralysis of the biceps, of the semitendinosus and semimembranosus muscles, of the movements of the foot upon the leg, as well as of the different parts of the foot. The foot dropped and this interfered with the gait. Walking, however, was possible. There was complete anæsthesia in the distribution of the great sciatic nerve. The muscles were atrophied and flaccid and the temperature of the affected leg was less than that of its fellow. Faradic sensibility was diminished in the flexor muscles already named in the posterior part of the thigh, and was abolished in the peroneal muscles, in the extensors of the toes, in the tibialis anticus, and in the diverse muscles of the foot. This case is related in great detail by Annequin and is a valuable contribution to the results of injury of the sciatic nerve. For purposes of reference it is by far the most valuable case on record. Bouilly[106] observed a case of contusion of the

sciatic nerve in a man, aged fifty-five, who fell while asleep, violently bruising the left buttock. There was neither luxation nor fracture. The accident was followed by paralysis of the limb, with atrophy of the muscles of the buttock, of the thigh, and of the leg, and other symptoms of neuritis. The author reviews the history of other cases of contusion of the buttock, and ascribes many of the symptoms of these cases to a lesion of the sciatic nerve. He believes, however, that while injuries to the buttock are not rare, contusion in these cases of the sciatic nerve is far from being common. This paper of Bouilly is also a minute statement of nerve injuries resulting from traumata about the buttock, and is a valuable contribution to both the surgery and the pathology of those cases in which the sciatic nerve may be involved.

The sciatic nerve has also been involved in tumors such as sarcomata, myxomata, and fibromata. Coupland and Balding[161a] report the case of a man, aged thirty-four, who had a large fibrosarcoma, involving the lower six inches of the sciatic nerve. The patient had had pain in the leg for three years; for one year he had noted a swelling at the back part of the thigh, a short distance above the popliteal space. The pain had been so severe as to interfere with his work. Coupland excised this tumor. At the operation it was discovered to be oblong or spindle shaped, four to five inches long and one and a half inches in width. It was completely ensheathed in the sciatic nerve, and in order to remove the tumor it was necessary to divide the nerve both above and below it. Five inches of the nerve were thus exsected. This tumor returned after the operation, and a secondary growth appeared in the anterior mediastinum, and eventually assumed immense proportions, almost completely filling the left side of the thorax. The tumor was a fibrosarcoma, evidently of a very malignant character.

Camperon and Cornil [162] reported a case of tumor of the sciatic nerve in a man aged twenty-two. Within one year after its appearance it grew rather rapidly. It caused no pain except on pressure, and no distinct motor symptoms, except a slight affection of the gait, with trailing of the point of the foot. The tumor was found to be incorporated within the left popliteal nerve. This nerve was profoundly altered. The neoplasm seems to have infiltrated the nerve, and to have been a fibromyxoma. Cornil made a minute histological examination of this growth. The new tissue grew between the nerve fasciculi composing the nerve trunk. The sheath of Schwann of individual nerve fibres presented a proliferation of the nuclei. Cornil says that the intrafascicular connective tissue had developed in a morbid way and constituted the growth. It was

not therefore a true neuroma—*i.e.*, it did not spring from the nerve tissue proper, but from the connective or mesoblastic tissue of the nerve trunk. This observer seems to think that it was unique, although with our present knowledge it does not appear to have presented any extraordinary histological characteristics. It was remarkable, however, in this case that the axis cylinders were preserved; and this state corresponds with the clinical fact that the sensory and motor functions were but little, if at all, affected.

Page[164] observed a case of cystic tumor of the sciatic nerve in a man aged thirty-six years. The tumor had been of four years' duration. On admission to the hospital there was no loss of sensation. At the operation the tumor was found to be entirely within the substance of the great sciatic nerve, the sheath of the nerve constituting, as it were, a capsule. In this case there had been some difficulty in determining whether or not the tumor was an aneurysm of the femoral artery; especially as, after puncturing it, distinct pulsation had been felt. The tumor was a sarcoma, which had evidently undergone cystic degeneration. In this case also it is noteworthy that no loss of sensation was observed. This would seem to indicate that the nerve fibres had remained intact. De Schweinitz[165] reports a case from Ashhurst's clinic, of a tumor which had developed on the stump of an amputated thigh in connection with the sciatic nerve. It was a fibroma and the chief symptom was extreme pain. Entire relief followed excision, which was performed by Ashhurst.

Tiffany[166] reports a case of a ship carpenter, aged sixty years, who had, scattered over his body, many tumors of fibroma molluscum. These had been present for many years. The first symptom of disorder in the sciatic nerve was drooping of the toe, causing a tendency to trip, and followed by absolute loss of power in the foot and leg, without pain. Later pain appeared, extending downwards from the middle of the thigh. At this time, also, a lump was noticed at the back of the thigh. At first paroxysmal, the pain tended gradually to become persistent. The tumor was larger than a closed fist. It was fusiform in shape, the long diameter corresponding to the direction of the sciatic nerve. It was freely movable from side to side, but less so from above downwards, and any position that tended to stretch the sciatic nerve made the tumor less movable. Pressure upon the tumor caused pain in the whole trajectory of the sciatic nerve. The electrical reactions were altered in the sural and peroneal groups of muscles and in the anterior tibial. The growth was found on removal to be an encapsulated tumor of the sciatic nerve, entirely unconnected with surrounding structures.

Marchand [167] also reports a case of cystic sarcoma of the sciati

nerve. It occurred in a man aged thirty-eight years, who had perceived for five years a swelling situated deeply in the posterior part of the left thigh. As in most cases, there was for a long time no spontaneous pain, but acute pain was caused by any accidental violence to the tumor. After a very slow course of three years the patient began to have some more active symptoms. Pain then began, with disturbance of the motor functions. Eventually the pain became severe and quite interfered with progression. This tumor is described as oval in shape, as in most other cases reported. The sciatic nerve is described as entering it at its superior pole, then separating from it, forming a sort of plexus of the nerve fibres, occupying the semicircumference of the tumor on its posterolateral aspect. The nerve then became restored, as it were, into its two main branches, the internal and external popliteal, just below the tumor.

Other cases of tumor of the sciatic nerve are on record. In all the growth consists of proliferated connective-tissue elements, constituting a true tumor, usually sarcomatous in type. It springs entirely, as a rule, from within the nerve sheath and eventually forms a large oval or fusiform tumor. Through the new tissue the nerve fibres course, and in some instances the axis cylinders may even remain intact. This accounts for the fact that several years even may elapse before active symptoms of involvement of the nerve fibres appear. These tumors cannot be enucleated from the nerve structure. They present the appearance of an infiltrating mass in which the nerve fibres are hopelessly incorporated.

Allis [187a] has described the mechanism of injury of the sciatic nerve in dislocations of the hip. The accident occurs especially in dorsal luxation, in which the head of the femur may pass between the nerve and the tendon of the outer hamstring or biceps muscle. The injury may be so severe as to cause a prolonged neuritis, with partial or complete paralysis. Quain reported a case in which the head of the femur was forced beneath the obturator internus muscle, and the great sciatic nerve was stretched over the neck of the bone.

The *symptoms* caused by paralysis of the great sciatic nerve cannot be better shown than in the cases already cited, in which a portion of this nerve had been exsected for a tumor growing within its sheath. In Coupland's case, already referred to, a segment of five inches, including the morbid growth, was removed. The region of the excision was a few inches above the popliteal space, consequently above the division of the nerve into its two main branches. The condition of the limb at the end of six months was briefly described as follows: The knee was semiflexed, but movement of the joint was preserved.

There was no movement at the ankle-joint, the foot being inverted and toes extended. There was much muscular wasting of the leg, and walking, which was done with the aid of crutches, caused œdema of the leg and lower third of the thigh. Sensibility was retained on the front of the leg and dorsum of the foot, but was abolished on the back of the leg and on the sole. A slough formed upon the heel, but the ulcer cicatrized. An indurated mass, somewhat painful, persisted in the upper part of the cicatrix at the proximal end of the divided nerve. The preservation of sensation in this case, although not very definitely mapped out, was probably in the area supplied by the long saphenous nerve. The movement of the knee-joint was preserved in this case because the muscular branches of the sciatic nerve, which supply the flexors of the leg, are given off above the seat of the operation. In Tiffany's case[166] the nerve was excised just above its division into the two popliteal branches. Two and a half months after the operation there was absolute paralysis of sensation and motion as follows: Anæsthesia was complete in all the leg below the knee, except in the area supplied by the long saphenous nerve, which arises from the anterior crural. In walking the left knee was kept slightly bent, the quadriceps extensor muscle being, as it were, overactive in all movements. The bending of the knee was probably done to permit this muscle or group of muscles to gain a firmer purchase. The foot dropped and was perfectly flaccid from the ankle, but took a proper position when helping to support the body. Tiffany insists upon the fact that this patient relied largely upon and was guided by the quadriceps extensor muscle. He thinks that the muscular sense of these anterior thigh muscles compensated to a great extent for the loss of sensibility below the knee. In Marchand's case,[167] in which a cystic sarcoma was excised with a portion of the trunk of the nerve, including the two main branches of the nerve in the popliteal space, the symptoms four months after the operation were briefly as follows: The limb was the seat of an œdema somewhat similar in appearance to scleroderma; nevertheless the circulation did not seem languid, although the skin had a slightly reddened hue. Anæsthesia was complete in the posterior and external aspects of the leg. The skin of the foot, with the exception of the internal border and of the great toe, was likewise anæsthetic. On the internal aspect of the leg, on the contrary, the skin had preserved its sensibility. This preservation of sensibility corresponded exactly to the distribution of the internal saphenous nerve. The patient was able to walk, even easily. The extension of the leg was performed regularly. The vault of the instep was no longer maintained, and the sural muscles were completely paralyzed. The foot

became flat each time that it supported the weight of the body, and at the same time turned outwards just as in the valgus of flatfoot, which it resembled remarkably. There was simultaneous paralysis of the peroneal muscles, the sural muscles, the anterior and posterior tibials, and the extensors and flexors of the toes. This paralysis accounts for the effacement of the plantar arch and for the abduction of the foot. Marchand does not give further details, but evidently, from the muscles paralyzed, the patient had lost all power of motion of the foot and toes.

The symptoms just enumerated are those, of course, that are caused by total transverse lesions of the nerve trunk. Such lesions of the sciatic nerve are not likely to occur except as a result of an excision or wound, either accidental or surgical. Inflammation of the sciatic nerve does not usually act as a total transverse lesion, although it is conceivable that it might do so if of a very high grade and far advanced. Compression of the nerve trunk has been reported as caused by the patient sitting upon the hard edge of a chair or stool, but in these instances the lesion is not transverse and complete. In ordinary forms of inflammation or irritation of the sciatic nerve the symptoms are those of the disease commonly known as sciatica, which will now be described.

Sciatica.

Etiology.—Syphilis as a cause of sciatica has been recognized by the ablest observers of this disease. Ludovic Dubois[1] has given a full *résumé* of this subject in his very able thesis. The older observers, while recognizing various neuralgic affections due to syphilis, do not make special mention, as a rule, of sciatica. Gros and Lancereaux, Zambaco, and Lagneau, in their observations on nerve syphilis admit the existence of syphilitic sciatica. They claim that the symptoms of pain in the sciatic nerve may even exist without gross syphilitic lesions causing compression. Dubois also admits the existence of syphilitic sciatica as the so-called essential neuralgia due to the syphilitic diathesis. Sometimes, however, these pains are the symptoms of compression, and sometimes they may be caused by the presence in the interior of the nerve of nodules, which are in reality gummata. Dubois' general conclusions are as follows:

Sciatica due to syphilis is more frequent than is supposed. It manifests itself at diverse periods of the evolution of the disease, and arises from various causes. In the period of secondary activity it is probably due to a virulent subacute neuritis, which can be compared to the sciatica caused by various other infections, as

by purulent infection, and by the poison of typhoid fever. He claims that syphilitic sciatica presents symptoms so characteristic as to permit it to be distinguished clearly from any other kind of sciatica. These symptoms are very acute—continued pains with nocturnal exacerbations, with trophic lesions and muscular atrophy, associated with other secondary symptoms of syphilis. In the period of tertiary activity the sciatica is caused by exostoses or gummata, which compress the nerve, or possibly by gummata which developed in the interior of the nerve trunk. Finally, syphilitic sciatica is always amenable to specific treatment, appropriate to the period during which it develops. In this connection it is important to call attention to the necessity of distinguishing true sciatica of syphilitic origin from the fulgurant pains of posterior sclerosis of the spinal cord due to syphilis. In the latter instance the pains are bilateral, and are associated with other symptoms of locomotor ataxia, especially abolished knee jerks, incoördination, and optic atrophy.

Sciatica is no doubt associated in some cases with the rheumatic and gouty diathesis. In very many instances, however, these constitutional states can only be suspected. In the vast majority of these cases patients suffering with sciatica do not present marked symptoms either of constitutional gout or of rheumatism. It seems, indeed, that the tendency to ascribe inflammation of the sciatic nerve to either gout or rheumatism arises from the difficulty or inability of the observer to attribute it to any other cause. Certainly very many cases of so-called idiopathic sciatica, in which a gouty or rheumatic diathesis is suspected, do not respond to appropriate constitutional treatment for these diseases.

The influence of exposure to cold as a cause of sciatica cannot be denied. This, I think, is true in cases especially of persons whose general health is in any way impaired. Thus men who are underfed and overworked and who are then exposed to cold, especially in association with dampness, are liable to these attacks. Excessive use of alcohol is probably a predisposing factor in such cases. Finally, it cannot be denied that many cases of sciatica occur in which the causation is altogether obscure. I have known it to occur in a perfectly healthy woman, in whom no satisfactory cause could be traced. In another instance, a physician in the prime of life, apparently in perfect health, became a martyr to sciatica for two years. The only cause that was suspected was rather high living with a somewhat too free indulgence in alcohol. No symptoms attributable to gout were observed.

Malaria has been supposed by some, in the absence, however, of

very conclusive evidence, to be a cause of sciatica. Such cases, however, do not yield, as a rule, to treatment by quinine.

Lead and the other metallic poisons have not been satisfactorily shown to be causes of sciatica.

Symptoms.—The chief symptom is pain coming on either abruptly or gradually with mild paroxysms. Occasionally it is noted in the beginning only when the patient makes some unusual motion or effort. Thus in one case of which I have knowledge, the patient for some weeks had noticed a sharp pain in the calf of the leg on sneezing. This was the beginning of a most painful and obstinate attack of sciatica. In other cases the patient feels these initial pains on suddenly rising, or bending the leg, or especially on forcibly bending the thigh or throwing the thigh muscles into rigid action, as on attempting to lift a weight. This fact often leads the patient to regard some particular movement or effort as the cause of the disorder; and this, I am sure, is often if not always an error. The pain under these circumstances is merely an indication that the nerve trunk is the seat of a beginning inflammation, and the pain is excited by the sudden violent action of the muscles which either extend or press upon the nerve trunk. Thus a forcible action of the pyriformis muscle might very readily cause sufficient pressure to excite pain in a hypersensitive nerve, whereas a healthy nerve would be entirely insensible to this action. In the early stages of the disease the pain is usually paroxysmal. Not infrequently, however, it is insidious, more or less constant, of a dull aching character, and prevents the patient from keeping the leg in a comfortable position. As the disease advances the pain becomes much more severe, and may be of a sharp, tearing, or lancinating character, especially intense on movement. Coughing and sneezing usually aggravate it, and in some instances these actions are much dreaded by the patient. The pain of sciatica is usually felt along the whole course of the nerve, extending even into the foot and toes. There are certain points, however, which are especially sensitive. These are at the exit of the nerve from the pelvis, in the popliteal space, at the head of the fibula, in the calf, and occasionally on the back of the foot. The trunk of the nerve is usually exquisitely sensitive on pressure. This furnishes an important aid to diagnosis. Thus firm pressure on the nerve trunk where it emerges from the pelvis, midway between the tuberosity of the ischium and the great trochanter, seldom fails to make the patient wince and to cause him to utter an exclamation of pain. The whole trunk of the nerve in its course down the back of the thigh may likewise be painful on pressure. In the popliteal space both popliteal branches may also be hypersensitive.

In addition to pain, other affections of sensation may be present. The patient may have tingling or burning sensations, especially in areas where the terminal sensory filaments are distributed. Anæsthesia is not common, although it is not unobserved. It usually requires careful tests for its determination. With these tests some areas of distinct anæsthesia, or especially of retardation of sensation, may be detected below the knee.

As so often happens in inflammation or irritation of nerve trunks, the motor fibres are not nearly so seriously involved as the sensory. Distinct paralysis of the muscles supplied by the sciatic nerve is not usually observed in sciatica. Impaired or inhibited movement due to pain is not uncommon, but this should not be mistaken for a true paralysis. In cases of long standing, however, the muscles of the calf and leg may become somewhat flabby. This is probably as much from misuse as from any real degenerative action in the muscular fibres. Reactions of degeneration are not observed, as a rule, in sciatica. This is according to my own observations and the reports of others, but of course I would not deny the possibility of impaired electrical reaction in cases of far advanced sciatica in which the motor fibres of the nerves might become impaired. Trophic lesions are not often seen in sciatica; at least I have not observed them nor seen reports of them. Gowers says that herpes may occur, but he gives no instance.

The *duration* of sciatica varies greatly. It is only too apt to be a most obstinate affection. Cases extending over two years are not unheard of. Relapses may occur, and thus the duration of the case may be much protracted. In many severe cases, after improvement begins, there is a long term of mild chronicity. The patient, while relieved of intense pain and severe paroxysms, is still conscious of more or less soreness and tenderness in the nerve trunk. While this condition lasts relapses are to be feared. On the other hand, some mild cases may last only a few weeks, although in my observation such mild cases are extremely rare.

The *pathology* and morbid anatomy of sciatica have been subjects of much dispute. Luria,[104] who has reviewed the history of this disease from the time of Galen, shows what an obscure idea, or rather what profound ignorance, has characterized the writings of many authors on this subject. The opinion of Galen that sciatica is only a species of gout and has for its cause a superabundance of some humor in the blood and articulations, and that this humor distends the nerve sheaths, thus causing pain, was followed for many centuries by various writers. Yet some of these ideas of the humoral pathologists are strangely in accord with those towards which modern

speculation and observation are tending. Thus Cotugno, who according to Luria was the first to give a good description of sciatica, wrote that it was the deposit of a large amount of irritating matter in the sheaths of the sciatic nerve that occasioned sciatica. This irritating matter can cause an inflammation of the sheath, and it is in this way that the most painful forms of sciatica are caused. Descot,[170] who wrote in 1825, had distinct ideas on the subject of sciatica. He wrote that neuralgia, at least in most cases, was only an intermittent or remittent neuritis. Valleix, however, in 1841, claimed that anatomical researches had not enlightened us upon the nature of sciatica, and he concluded that it was demonstrated that sciatic neuralgia, just as other forms of neuralgia, is due to a state of the nerve which cannot be discovered by the scalpel, and which is only manifested by a "lesion of the functions." This extraordinary opinion seems to have been followed by many subsequent writers, so that it has become rather the custom to regard sciatica and neuralgic affections of other nerves as, in many instances, due to some quite incomprehensible alteration of functions without a definite anatomical basis. Thus Marchesseux in a thesis denied the inflammation of the nerve in sciatica. Handfield-Jones, in 1864, taught that sciatica, except of recent rheumatic origin, was not inflammatory, but that it was allied habitually to a feeble state of health. Thus opinions varied as to the true underlying process in sciatica. The clinicians, such as Lasègue, Trousseau, Landouzy, and others, were apparently reluctant to admit a gross organic lesion as the basis for sciatica. Thus the disease continued for a long time, especially in France, to be regarded as a "neurosis," *i.e.*, a disease without a recognized pathology. The history of the course of opinion upon the subject of sciatica is given in detail in Luria's thesis.[169] In 1878, however, Furnet[171] published the results of his researches upon sciatica, and expressed his opinion that this disease is due in the greatest number of cases to a neuritis. He published one of the earliest reports of an autopsy in sciatica. In this case the patient had died of acute tuberculosis of extremely rapid course. He had been suffering with sciatica. At the autopsy there was found an inflammation of the sciatic nerve. Furnet cites from other observers proof that the nerve and its popliteal branches were more voluminous than normal and that the vessels were dilated and distended. Martinet, quoted by Luria, reported an autopsy in the case of a man who had suffered with sciatica of the right leg during the last five days of his life. Sound at its origin, the sciatic nerve became reddened at its point of emergence from the pelvis, and pus was disseminated between the nerve fasciculi. In still another case this observer found

the sciatic nerve reddened in color from an extravasation of blood between its fasciculi. In a man who, after a forced march, was seized with very severe pains in the posterior part of both thighs along the course of the sciatic nerves, Martinet found the nerves notably hypertrophied, hard, injected with blood, and presenting an infiltration of bloody serum between their fasciculi.

Gendrin, also quoted by Luria, observed in many subjects who had presented during life the symptoms of sciatic neuralgia, a red or violaceous color and a vascular injection of the sciatic nerve, with small clots of blood distributed in its interior, and hypertrophy of the nerve, and its degeneration into a soft or spongy material.

I can add to these records an observation of my own in the Philadelphia Hospital. In this case a man, suffering with a severe form of sciatica, died after a short illness from some intercurrent pulmonary affection. At the autopsy the sciatic nerve was found swollen, engorged, and distended with a bloody serum, its sheath thickened, and its fibres macerating, as it were, in the products of inflammation.

From these observations, which might be supplemented by others taken from the literature of this affection, there can be no doubt that sciatica, at least in its severe forms, is only another name for neuritis; and yet the disease presents some problems from this point of view that are not altogether simple. For instance, the chief and often the only symptom in sciatica is due to irritation. This is pain. Paralysis or abolition of function of either the sensory or motor neurons is but rarely observed. There may be at times in some cases, it is true, some areas of anæsthesia, showing that the axis cylinders of the sensory neurons have lost the power of transmitting impressions. Paralysis of the motor neurons, however, is not often if ever observed. The inference to be drawn from these facts seems to be that some irritant, just as was taught by the old humoral pathologists, is circulating within the nerve sheath, and that this irritant, while sufficient to excite an extremely painful reaction in the sensory neurons, is not in the vast majority of cases sufficiently virulent to destroy the axis cylinder in its continuity or even to vitally impair its conducting power. This, we know, may occur in some other nerve trunks as well as in the sciatic. It is not, however, the rule in any instance in which a high grade of inflammation is present in a nerve. Thus in the neuritis due to alcohol, while pain is often a common and early symptom, it is soon associated with loss of muscular power and with various degrees of anæsthesia. The explanation of this difference may possibly be found in the fact that in alcoholic multiple neuritis the seat of inflammation is farther towards the peripheral nerve endings; whereas in sciatica the inflammation is in

the main nerve trunk. In the former case the axis cylinders are probably more exposed to destructive and degenerative processes for two reasons: first, because they are not so well protected in the sheaths of the nerves; and second, because in these situations they are farther removed from their trophic centres—*i.e.*, the cell bodies of the neurons—either in the anterior cornua of the gray matter of the spinal cord or in the ganglion on the posterior root. A third reason may possibly be found in the fact that the capillary circulation is probably richer and is certainly spread out over a wider area in the periphery. Thus the delicate terminal arborizations of the neurons are more exposed to a completely destructive process, caused by the circulation of a poison in the blood, than are the main trunks of the axis cylinders high up within the nerve sheath. Whatever may be the explanation, the facts are as stated, and we must conclude that a high degree of inflammation may exist in the trunk of the sciatic nerve without producing marked symptoms of pressure upon, and much less of destruction of, the axis cylinders.

The seat of this active inflammation in sciatica is usually in the main nerve trunk, probably beginning about at the point of its exit from the pelvis. This was shown in one of Martinet's cases quoted above. The nerve was sound at its origin, but became inflamed as it emerged from the pelvic cavity. From this point the inflammation usually extends downwards, and may extend with diminishing virulence even into the trunks of the two main branches of the nerve in the popliteal space. This was practically so also in my own case, above referred to. The appearance of the nerve, as already indicated, is that of a neuritis, not only involving the nerve sheath but also the interstitial tissue. Extravasation of serum and blood between the fasciculi has been observed in most reported cases. The nerve trunk itself is markedly altered in appearance. It is usually swollen, reddened or purplish in appearance, and in recent cases rather soft to the touch; in long-standing cases the nerve sheath may be thickened, and the nerve then gives the impression of increased hardness to the touch. Gowers claims that the changes chiefly involve the nerve sheath. If this is so, it is an additional reason for the comparative exemption of the motor and sensory neurons from destruction and even from pressure. From the reports of observed cases, however, it by no means appears that this is always the case, because the interstitial tissues seem to be the seat also of the morbid process, and the extravasation of blood and serum undoubtedly occurs between the fasciculi of the nerves. Some observers have reported the presence of pus within the nerve sheath. The minute microscopical appearances of the nerve fibres have not been reported in most

observed cases with which I am familiar with sufficient exactness to admit of conclusions. As said already, however, it seems almost certain from clinical data that in the vast majority of cases few if any of the axis cylinders are destroyed. In cases of complete division of the sciatic nerve, such as have been referred to already in this paper, the process of degeneration and repair undoubtedly proceed just as has been described in the section on the general pathology of nerves (see page 42). Park's case,[166] in which complete rupture of the sciatic nerve was followed, after suturing the nerve ends, by complete restoration of motor and sensory functions within two weeks, must be accepted with great reservation. We know already from Stroebe's researches that the process of regeneration in divided nerves is extremely slow. It is inconceivable that the divided axis cylinders can reunite so promptly and so accurately, each end to its appropriate fragment in the distal portion of the divided nerve, that complete restoration can occur in such an incredibly short space of time as two weeks.

The *diagnosis* of sciatica is, as a rule, not difficult. The peculiar onset of the disease; the characteristic pain along the course of the nerve; the sensitiveness on pressure of the nerve trunk; the usual exemption from paralysis of the motor and sensory fibres—all indicate very clearly the nature of the affection. There are some affections, however, that may very readily be mistaken for sciatica. The chief of these are various organic lesions within the pelvis. These have already been clearly indicated in the section on diseases of the sacral plexus. To recapitulate very briefly, I may mention especially abscesses within the pelvis, diseases of the uterus and its appendages, large fæcal accumulations, and malignant growths of the rectum. Abscess within the pelvis, especially when pointing at the great sacrosciatic foramen, has simulated the pain of sciatica. In such a case thorough exploration of the pelvis, and especially the detection of diseased bone, either of the vertebral column or of the pelvis, and a study of the temperature range, will usually determine the true nature of the disease. Inflammatory affections of the uterus and of the periuterine connective tissue, especially following the puerperium, can usually be determined by the history of the case and by a thorough exploration of the pelvis. In my own case of small ovarian cyst causing pressure on the sacral nerves, an examination per vaginam at once cleared up the diagnosis. In such cases the pain is usually not limited to the course of the sciatic nerve, but radiates about the groin and buttock and even towards the hip. Disease of the rectum can be determined, of course, only by a careful exploration of the part. Obscure cases with sciatic symptoms, especially in women,

are never properly studied without a pelvic, and in particular a rectal, examination. This point cannot be too strongly emphasized. Hip-joint disease or rheumatism about the coxic articulation would not likely be mistaken for sciatica. An examination of this joint by the methods well recognized by orthopedists should establish the diagnosis. Appendicitis may cause obscure symptoms, especially pain deep in the pelvis, but this pain does not, as a rule, follow the course of the sciatic nerve. Exact physical exploration in these cases seldom fails to determine induration and sensitiveness in the region of the appendix.

The *treatment* of sciatica requires, as a rule, great patience, tact, and discrimination. This disease has baffled the skill of therapeutists for many generations, and the history of its treatment is one of the most interesting in clinical medicine. It has always been a disease which has invited and received heroic treatment, and some of this treatment has stood the test literally of ages. Although founded upon empiricism and a complete ignorance of the true pathology of the disease, much of the old-time treatment for sciatica was admirably adapted to the exigencies of such a deep-seated organic affection. It cannot be said, indeed, that in modern times we have advanced very far beyond the older therapeutists, and it is even possible that we may never be able to advance beyond some of the wise and efficient plans of treatment which they advocated. As, however, all rational or scientific therapeutics is founded upon an accurate knowledge of the conditions to be treated—*i.e.*, the pathology of the affection—it may be that, when we come to recognize more fully the inflammatory nature of sciatica, we may treat it more radically by surgical procedures.

It must be said, in the beginning of a discussion of this subject, that there is no panacea for sciatica or no one plan of treatment that is adapted to all cases. I shall attempt to give some of the results of my own observations, as well as to review briefly the subject from the standpoint of the accumulated experiences of others.

Among the methods historically noteworthy, acupuncture may take the first place. This plan of treatment is very ancient, and, according to common report, has been used by the Chinese for sciatica from time immemorial. They employed for this practice very fine gold or silver needles, made with a small handle so as to be able to impart to the instrument a rotary movement. According to Luria, the method of the Chinese is to sink the needle almost up to the seat of pain, observing care not to prick the nerve itself. This, however, is not the idea which I have always entertained of the Chinese method of acupuncture. I have supposed that the object of this method was

to penetrate the nerve sheath, and I have supposed that its efficacy depended upon the fact that, although the punctures must be very minute, they still were efficacious for draining away some of the fluid products of inflammation. Cloquet presented to the French Academy of Sciences, in 1824, a memoir of the results obtained by him in a great number of patients. His conclusions, quoted by Luria, are as follows: First, that acupuncture acts generally upon the pains, whatever may be their underlying causes; second, that some of these pains disappear never to return; others reappear after a variable time, but always more feeble; and finally some of the pains are not modified. Cloquet speaks as though the beneficial effects may be due to an inflammation excited by the needle. This observer cites cases cured promptly by this means. He possibly overestimated the value of the treatment. In one case of my own, a kind of acupuncture was inadvertently practised by the use of the hypodermic needle. The patient, a lieutenant of police, had suffered for many months with the worst form of sciatica. The pain on pressure was greatest at the point of exit of the nerve from the pelvis. A syringe was used a few times in succession for the purpose of injecting morphine deeply in the neighborhood of the nerve trunk. It is even possible that one of these injections may have been made directly into the sheath of the nerve. Unfortunately, owing to some laxity in aseptic precautions, these injections caused abscesses deep in the buttock. These abscesses required to be freely excised and continued to discharge pus for at least two weeks. The therapeutic effect of this accident was on the whole beneficial. The pain was assuaged, and never again returned in its former severity. A cure, however, was not effected. The patient continued to suffer for many months subsequently with a chronic, although a milder, type of the disease. It is needless to say that, while this plan of treatment is not recommended, it probably illustrates, by an extreme example, the method of action, to a certain extent, of acupuncture. In case acupuncture is used, very fine needles are to be preferred, and they should be used with every aseptic precaution. They need not be of either silver or gold. The emphasis which has been put upon this subject by some writers leads one to suspect that probably a mental impression has been sought and obtained in many of these cases, and that after all we have here a form of suggestive therapeutics. Certainly there is no reason why a clean steel needle should not be as good as one of silver or gold.

According to Luria, Cotugno or Cotunnius, whose treatise on sciatica appeared in 1770, instituted the treatment of sciatica with blisters. This observer taught that the blisters should not be

applied indifferently along the whole extent of the nerve, but upon the part of the nerve placed most superficially; consequently he cautioned against the application of a blister, so commonly made, over the nerve where it issues from the pelvis, since it is here placed deeply beneath the gluteus and other muscles and tissues. He advised the application of the blister by preference over the trunk of the external popliteal branch of the sciatic nerve at the superior and external aspect of the leg. From my own observations, the action of blisters in sciatica is often neither prompt nor efficacious. The suggestion of Cotunnius, however, that the blister be placed at the most superficial point of the nerve, or rather of one of its main branches, is one that is not unworthy of consideration. It may be, as this author suggests, that the reason for the frequent failure of this plan of treatment is because the blister is nearly always applied over the point of exit of the nerve from the pelvis. In most persons, and especially in those who are at all corpulent, the skin and fat and connective tissue over this portion of the nerve are very thick and dense, and must as a consequence offer much resistance to the proper action of a blister upon the inflamed nerve. Although the seat of most active inflammation is probably above and remote from the external popliteal or peroneal nerve, yet the trunk of this nerve approaches so closely to the surface that the benefit derived would probably be greater than it would be in case the blister were placed over the seat of greatest irritation. When a blister is used the effect should be long maintained—that is, it should be kept as an open, running sore. Probably one reason for the failure of this treatment is that too much is expected from one application. The blister is allowed to heal up too promptly; disappointment results, and the treatment is abandoned. To maintain this treatment properly, it is usually necessary to apply a blister every few days. By this means an open or suppurating sore is secured, which keeps up a constant action, not unlike that which results from a superficial burn. This plan of treatment by successive blisters was insisted on by some of the older observers, especially in France. The treatment is probably best adapted to mild cases, and especially in the early stages. In chronic cases I have not tried it, but I suspect that it would not act so efficaciously as the hot iron, which will be referred to presently. Among the curiosities of medicine may be mentioned here the cauterization of the end of the ear of the affected side with the hot iron; also the catheterization of the Eustachian canal, both recommended in olden times for sciatica.

The treatment of sciatica with various caustics is also very ancient. It is mentioned by Sanctorius, according to Luria, and also by Riverius, who conceived the fanciful idea that as sciatica was produced

by a catarrh descending from the brain, the caustic should be applied to the occiput! The treatment by caustics was especially recommended by Trousseau. Various agents were used but these need not be enumerated. The mode of action of caustics is evidently the same as that of continued vesication or of the hot iron. Sulphuric acid is especially mentioned by the older observers.

The treatment of sciatica with the hot iron is said to have been employed by Albucasis in 1106. He made three lines with the hot iron along the course of the nerve at a distance from each other of about the thickness of the finger. Jobert, speaking of this treatment, claims that it allays the pain, but he seems to incline to the belief that it is necessary to make a deep impression, because if applied lightly it does not give the same results. He suggests that the iron should be very hot, a recommendation which is in accord with that of contemporary observers. Luria inclines to the belief that the hot iron is not a sovereign remedy. My own observation of this treatment has led me to believe very firmly in its efficacy. In one very severe case of sciatica which had lasted for nearly two years and had proved most rebellious to all forms of treatment, I saw the use of the hot iron effect a radical cure. In this case it was applied in four places along the course of the nerve by a Paquelin cautery raised to a white heat. Each spot burned was slightly more than one inch in diameter and was immediately treated before the patient recovered from the anæsthetic with an application of pure carbolic acid. This treatment made sores which were a long time in healing. Sloughs separated and open running sores were thus maintained for several weeks. This heroic application of the hot iron, however, is not always required and is not in accord with the recommendation of all neurologists. Thus Weir Mitchell recommends a series of very light touches of the actual cautery, raised to a white heat, along the course of the nerve. By this method little more than the epithelium of the skin is destroyed and the resulting sore heals promptly and leaves little, if any, scar. In one case in a woman I recently had the most gratifying results from this slight application of the actual cautery. The case, however, was not so severe and chronic as the one referred to above. While I believe, in the main, that a light touch of the iron is all that is required in many cases, yet it is possible that in very chronic cases a deeper impression is beneficial. A good plan is to make a series of light impressions at first. If these are not efficacious in the course of a few weeks, a second and deeper application of the iron should be made. This treatment, in my observation, seldom fails to have a prompt palliative action upon the pain. It will not, however, always cure.

In one case in which three rather deep, sloughing sores were produced, the patient, although relieved for a time, gradually experienced a return of all his former symptoms, but in a milder form. The degree of heat to be employed in these cases is not a matter of indifference. The best result is obtained when the iron is raised to a bright cherry red or even white heat. As the skin rapidly abstracts the heat, this result cannot be well obtained by an iron or a glass rod heated in a flame. The Paquelin cautery, which admits of a more or less constant maintenance of a white heat, is by far the best instrument to use. Even with this cautery, however, the abstraction of heat is rapid and the point is very likely to be reduced to a dull red. It is therefore better to make repeated touches. Thus as soon as the degree of the heat falls upon application to the skin, the point of the cautery should be momentarily lifted and allowed to resume its full glow. Thompson recommends the use of a glass rod. This can be heated to a white heat very rapidly in the flame of a spirit lamp, held very close to the patient's skin. Thus instantaneous and repeated applications are made before the degree of heat is materially lessened. When the Paquelin cautery cannot be secured this use of the glass rod is probably preferable to the use of the iron.

The reputation of electricity in the treatment of sciatica, as in that of many other diseases, has been subject to singular fluctuations. It has enjoyed in the past a much greater reputation than it maintains at present. It is an old method of treatment, according to Luria, having been recommended by Cotunnius in his monograph on sciatica more than a century ago. Later, the illustrious Duchenne was a firm advocate of this treatment. He claimed that faradization of the skin can cure sciatica and gave details of its methods of application. The popularity of the two currents has varied according to individual observers. Althaus, following Duchenne, claimed that faradism could cure sciatica. Berger, quoted by Luria, made the astonishing claim, in 1872, that he had obtained good effects in at least seventy-three cases of sciatica, all of which had been unsuccessfully treated by other methods, including the constant current. On the other hand, Renault, Gibney, and De Watteville recommend the use of the constant current. In my experience electricity by either current has proved of very little value in sciatica. The depth at which the nerve trunk is placed beneath the skin and connective tissue and fat on the back of the thigh, in many patients, renders it doubtful if much of the current as it is ordinarily applied reaches the nerve. The choice of currents is not a matter of indifference. The constant current should be employed, especially if the symptoms are acute. The method of applying the current should be as follows: A flat sponge

electrode is held firmly over the sacrum or over the buttock of the affected side. A smaller sponge electrode is then used to make firm and deep, but not too painful, pressure over the more accessible portions of the nerve. Probably the best effect is obtained and more of the current caused to flow through the nerve by holding this sponge deeply in the popliteal space with the leg slightly flexed at the knee. The current strength should be regulated somewhat according to the tolerance of the patient. In acute cases in which the pain is very severe it will usually be found that the patient cannot endure a strong current. It is impossible to prescribe the current strength according to the number of cells employed. These vary in their electromotor force and the resistance of the skin and other tissues varies in different people. If pain is much intensified during the application of the current, it will be better to desist. The positive pole, as being theoretically the sedative pole, should be the one employed upon the nerve trunk. Sudden shocks, as by making and breaking the current while the sponges are applied to the skin, should be avoided. If the faradic current is employed, the method may vary slightly from that just given. The two sponges may be of equal size and they may be applied by lightly stroking the skin on the back of the thigh and over the muscles of the leg. By this means the muscles may be made to react gently. But violent muscular contractions with a strong current should be avoided. De Watteville[1"] recommends electropuncture of the nerve, but only by an expert operator familiar with the electrolytic effects of the current. This seems to be a modification of the time-honored acupuncture, but it is a method with which I am not familiar. This authority also speaks of galvanization of the nerve by means of a rectal electrode. By this means the current could undoubtedly be brought into closer contact with the affected nerve than through the skin. The best time to apply electricity in sciatica is probably towards the end of the attack. At this time some of the chronic symptoms, as persistent soreness of the nerve and some flabbiness of the muscles, are likely to be present. These seem to be benefited, in some cases, by the use of one or the other current. The muscular nutrition is probably best restored by the faradic current.

Hydrotherapy has been much praised, especially in France and Germany, for the treatment of sciatica. In this country, unfortunately, this method is too little employed, not only in this but in many other diseases. The fact that extravagant and incredible claims have been made for it should not close the eyes of American practitioners to its importance. Lagrelette[1"] confidently claimed that he had found in hydrotherapy a method of sure cure for sciatica.

The method adopted abroad is to subject the patient to the pack, which causes copious sweating. This secures, according to Fleury, an energetic revulsion upon a very large surface of the skin. Whatever the theory, the method is undoubtedly a valuable one. In one case I knew an active daily treatment by the ordinary Russian bath to be followed by the most beneficial effects. In this case the patient, who was almost incapacitated for walking, was taken to the establishment and lived there for a month. The difficulty in securing this treatment arises in many cases from its inconvenience and expense. The Turkish bath cannot be obtained outside of our large cities and towns. Many of our hospitals, unhappily, have not the facilities for a proper use of the bath. Still, cold showers and copious sweating with a warm pack can be obtained even under ordinary circumstances. In some cases, if it is not practicable or desirable to give the patient full bathing, a local application of alternate hot and cold water can be made to the limb. The leg, for instance, can be swathed in flannels wrung out of water and thus a local and copious sweating can be procured. While this treatment is often beneficial, it is not to be compared in efficacy with the more thorough general treatment by baths. For patients whose means will admit, a sojourn in some special bathing establishment may be tried. Warm salt-water baths, as now obtained at many seaside resorts, might also be tried in obstinate cases of sciatica. Whatever plan of hydrotherapy is adopted it should have always one object, *i.e.*, the active revulsive effect referred to long ago by Fleury, and which is obtained by copious sweating. Finally, a caution should be uttered against building too high hopes upon this as upon any other treatment. Cases occur that will not yield promptly to hydrotherapy, or occasionally will not even be benefited by it.

A special method of treatment for sciatica, formerly much recommended by some, was with hypodermic injections of nitrate of silver. Luria devoted his thesis, already quoted, to a complete presentation of this subject. According to him, Luton of Rheims was the first to employ this method. Other substances, especially chloride of sodium, have been used, but the nitrate of silver has been the agent most employed. The idea seems to have been to substitute an inflammatory local action in the nature of a phlegmon in the neighborhood of the nerve trunk. The originator sought to obtain a focus of suppuration, which he evidently believed acted as a revulsive upon the inflamed nerve. Nitrate of silver, according to Luria, has the advantage of being energetic and of determining a phlegmonous inflammation of such a kind that the process is confined within a limited region, and hence is better under control. Moreover, its

action is not temporary, as is that of ordinary acupuncture, since the inflammation provoked by the injection persists and causes a profound local irritation of long duration. The phlegmon may terminate either by resolution or by suppuration. This injection of nitrate of silver is painful, but it is not unendurable. It is, moreover, less than the pain of the sciatica which it is said to relieve. Luton employed a solution apparently of about ten per cent. in a dose of ten, twenty, or thirty drops. He made the injections at the painful points, beginning at the higher level of the limb. He directed that they should be made deep in the tissue. Successive injections are made with gradually decreasing strength of solution further down the course of the nerve. Luria concludes that the injections of nitrate of silver can be employed with success for all forms of sciatica, but that they are especially successful in cases in which there is an active neuritis. No grave complications had ever resulted from these injections, as was proved in numerous observations. The superiority of this mode of treatment is due to the fact that the irritating solution is carried into the tissues in the neighborhood of the diseased nerve.

Nerve stretching has been recommended, and was at one time used quite extensively in the treatment of sciatica. The operation was first performed by Billroth, in 1869. At one time quite popular, it has now fallen rather into disrepute. Chauvel"[4] has defined this operation as one in which a nerve trunk is elevated upon a blunt instrument in such a manner as to obtain a real increase in its length. This increase or elongation of the nerve trunk is the object and the essential point of the operation. Some minor procedures, such as pulling and pressing the nerve between the fingers, crushing the nerve between the jaws of a pair of pincers, or sometimes a simple *degagement* of the nerve, are sometimes included under the one term; but as they essentially differ, both in their processes and results, they should be distinguished. One method recommended by some, is the subcutaneous elongation of the sciatic nerve. It is claimed that forced flexion of the thigh upon the pelvis causes an elongation of the sciatic nerve greater than that which can be obtained by a force applied directly to the nerve itself. In the operation for nerve-stretching the nerve is uncovered by appropriate incisions parallel to the course of its trunk. In case of the sciatic nerve the middle region of the thigh is preferred, because the nerve here lies more superficially than it does between the tuberosity of the ischium and the trochanter. The nerve is lifted from its bed by a blunt instrument and stretched. The amount of force to be applied to the nerve was formerly a subject of some discussion. From observations made upon the cadaver it seems that the sciatic nerve can resist greater force than can the other

nerve trunks. This is easily explained by its greater size. Symington noted that under a traction of one hundred and thirty pounds the nerve was ruptured six times at the point of application of the force and eight times it separated at its root. The nerve has less resisting power, according to Gillette, in old and cachectic individuals than in vigorous adults. Gillette, noting these various facts, constructed an instrument for the purpose of measuring the force applied to the nerve trunk for therapeutic purposes. This instrument he called an "elongator." The amount of force to be applied to the sciatic nerve should not exceed twenty kilograms. The operation of nerve stretching, while apparently simple and harmless, has been attended in a few instances with disastrous results. Thus Gärtner, in 1872, quoted by Chauvel, reported a case of a woman aged thirty-eight years who had violent neuralgic pains along the course of the nerves in the right arm. The nerve trunks in the brachial plexus were exposed below the clavicle and stretched. Some of these nerve trunks were noted to be visibly affected. On return to consciousness the patient found that the pain had disappeared and did not reappear. This patient died on the fifteenth day from hemorrhage from the jugular vein and by entrance of air into the vein. The surgeon claimed that this vein had not been wounded during the operation, but it seems incredible that the patient should have bled to death from it if it were not injured. Nussbaum encountered great danger from ulceration of the popliteal artery in a case in which he had stretched the sciatic nerve. The method of action of nerve stretching is not altogether clear. Much speculation has been wasted upon it. It seems, according to researches of Valentine, Marchand, and others, that there are distinct organic lesions caused by nerve stretching. Among these lesions were noted hyperæmia, capillary hemorrhages, and rupture of the axis cylinders; but, according to some, the sheath of Schwann remains intact. In cases in which permanent injury results, the nerve is contracted and presents a large number of degenerated fibres. The immediate results of nerve stretching, it is claimed, are temporary anæsthesia without motor disorder, in cases in which moderate force has been used. A more prolonged or even persistent anæsthesia, with paralysis of motion, and trophic lesions, occurs when great force is used. From these facts it seems that nerve stretching, by interrupting the continuity of the nerve fibres, causes paralysis of sensation, and hence acts to relieve pain. It is probable also that it may have an alterative effect on the inflammatory process, checking this and promoting absorption. Chauvel concludes that in cases of sciatica, if cure results, this occurs only a long time after the operation. As already said, this operation is no longer held in high

favor. It by no means insures a cure and it is not free from dangers, among the greatest of which are rupture of the nerve, rupture of a contiguous artery or vein, and permanent paralysis and anæsthesia of the limb.

In recent years S. Weir Mitchell[xiii] has recommended absolute rest of the affected leg. He secures this by not only putting the patient to bed but by extending the leg upon a long splint. This treatment is persevered with until the tenderness of the nerve has subsided. The time will, of course, vary in different cases. This is a rational treatment. The complete immobility of the inflamed nerve no doubt tends both to subdue the activity of the inflammation and also to promote the absorption of the products of inflammation with which the nerve sheath is distended. This treatment, of course, can be combined with other treatment, as, for instance, counter-irritation with the hot iron and appropriate medication and feeding. The patient thus secures the benefit of a systematic rest cure, and as many of these patients are not a little neurasthenic and broken down from the effects of long suffering and discouragement, this rest-cure is highly beneficial.

Mitchell lays great stress on the importance of bandaging the leg with flannel bandages in combination with the treatment on a splint. The bandage no doubt acts not only by putting the muscles perfectly at rest and thus relieving all muscular tension and involuntary movements which the patient may make, but also by preventing the transmission through the nerve trunk of all motor impulses. Any one who has seen the extreme sensitiveness of a patient with sciatica, not only to slight movement, but even to the slightest tactile impression upon the skin, will realize that the perfect rest and security obtained by a well-adjusted flannel bandage must be both comforting and beneficial. The bandage may be applied daily, and should, of course, be kept on all night, a precaution which is not always necessary in the case of the splint. An additional advantage, which the bandage secures, is maintenance of the circulation and warmth of the limb, and the promotion, possibly, of a slight but constant perspiration.

Mitchell also recommends very highly the application of cold in sciatica. This is done by means of an ice bag. The best form is the long spinal rubber ice bag. This may be filled with cracked ice, wrapped in a few layers of flannel, and kept constantly applied along the posterior aspect of the thigh. If it is applied, however, on the outer side with a roller bandage, it is not necessary to envelop it. Mitchell has obtained from this treatment with the splint, bandaging, and the application of cold the most satisfactory results.

The treatment of sciatica with drugs furnishes a long chapter in its history. Every known alterative and sedative drug has been used in the usually hopeless effort to combat this painful and obstinate affection. It cannot be said, however, that any known medicine has stood the test of experience. Sciatica is unusually rebellious to treatment by medicinal agents. The various alterative and constitutional remedies have been used *ad libitum*. In rheumatic and gouty cases the salicylates, iodide of potassium, the various salts of lithium, colchicum, and arsenic have all had extended trial. I have never been able to assure myself that they were beneficial. In cases of a distinct rheumatic history I might perhaps have some confidence in the salicylate of sodium, but this drug should always be combined with treatment by rest and some form of counter-irritation, preferably light touches with the hot iron. The preparations of lithium and colchicum are apparently of little value in any form of sciatica.

In cases of sciatica in which a malarial origin is suspected, the patient should, of course, have any advantages that may come from an antimalarial treatment. Many good authorities hold to the belief that sciatica is occasionally caused by the malarial poison. The practitioner, in fact, should not close his eyes to the fact that sciatica is probably a disease with many causative factors. Malaria, rheumatism, gout, and syphilis, and, in fact, any form of dyscrasia that depletes the blood, may lay the foundation for an attack of this disease, following slight injury which would not have affected a healthy nerve. Hence anæmic states, following profuse hemorrhages or infectious diseases, may have a direct causative influence. In cases in which a distinct malarial poisoning can be traced, the patient should be treated with large doses of quinine, combined with iron and arsenic.

The influence of syphilis in causing sciatica has already been indicated in this paper. This possible cause should be searched for with the utmost care in every obscure case presenting sciatic pains. In case it can be determined that the patient has had a primary sore or any secondary lesions of syphilis, he should have the benefit of a thorough antisyphilitic course. I have already indicated my preference for hypodermic injections of mercury in all early cases of nerve syphilis. When it is considered that the lesion of syphilis in the sciatic nerve may consist of gummatous infiltration within the nerve sheath, the reasons and the need for heroic treatment do not require to be urged. Injections of mercuric chloride, in doses of one-eighth to one-twelfth of a grain, may be repeated daily or every other day over a period of several weeks. Great care should be observed that these injections are aseptic, and salivation, if pos-

sible, should be avoided. In case no good results are obtained after two or three weeks of this treatment it may as well be stopped. It may be combined with large doses internally of iodide of potassium. While I have had no direct experience with sciatica of syphilitic origin, yet I believe that the possibility of this factor as a cause should never be ignored; and in case it can be determined to be present, the treatment herein indicated is urgently demanded.

The use of sedative drugs will always be demanded sooner or later in cases of severe sciatica. The precautions already urged in this paper, relative to the use of all such drugs, cannot be too emphatically repeated in this connection. The risk is not small that the patient will become dependent upon them, and will suffer correspondingly in his general nutrition and morale, if they are given with too free a hand. If the patient is treated with absolute rest, with his leg enveloped in a bandage and fastened on a splint, with the application of cold, or, in more severe cases, with light touches of the actual cautery, the need for anodyne drugs will usually be reduced to a minimum. At most it will only be necessary, as a rule, to give a few hypodermic injections of morphine. If this drug is used, it is better to give one full dose at bedtime with the object of securing a good night. These injections may be continued, according to the requirements of the case, for a few nights; but if the patient is thoroughly under control with the rest treatment here indicated, it will generally be found that the morphine may be discontinued in a comparatively short time. Of other sedative drugs the most efficient are antipyrin, antifebrin, antikamnia, and phenacetin. These may be given in doses of from ten to fifteen grains two or three times a day. If, after a trial of some days, they are found, as not unusually happens, to have little controlling effect upon the pain, they should be discontinued. Their routine use is too apt to become a habit with the practitioner, and as they have a more or less depleting action upon the blood, they should be used only for a definite object, and if that is not soon attained, they should be discontinued. Thompson[145] believed that atropine, conium, and ergotin may be of great service as adjuvants, but he discourages a main reliance upon them. Recently Mikhalkine[146] has recommended nitroglycerin in the treatment of sciatica when the usual remedies fail. It is given in the following mixture: Alcoholic solution of trinitrin (one per cent.), 80 gm.; tincture of capsicum, 120 gm.; peppermint water, 240 gm.; five or ten drops to be taken three times a day. Hunsberger[147] has recommended strongly aconitine for sciatica. As, however, he is obliged to give the drug for two months his results are not brilliant. Cocaine is undoubtedly an efficient anodyne in all forms of neuralgia, and acts satisfactorily in

sciatica. It should be injected deeply into the seat of greatest pain. As the danger of the cocaine habit, however, is always great from the therapeutic use of this drug, it should be used with the utmost caution and reserve by the practitioner.

Negro[17] has reported a series of cases of rebellious sciatica, treated by compression, all of which he claims ended in recovery. The procedure is as follows: The patient is placed on his face with his legs extended. Both thumbs are applied on the trunk of the nerve at its point of exit from the pelvis, and it is compressed with the greatest possible force. At the same time slight lateral movements are made upon the nerve trunk. This is done for fifteen or twenty seconds, and after an interval of twenty minutes' rest the procedure is repeated. Negro claims that the second application is much less painful than the first, and that after it the patient is able to walk, and that he may be free from pain for several hours or even a day. To effect a cure this procedure should be practised about six times a day every two days until recovery is obtained. His claim that he has thus cured one hundred and thirteen cases of "rebellious" sciatica is an extraordinary one, and should be received with caution. The compression acts in a manner somewhat similar to that of nerve stretching.

Finally a word must be said about the general nutrition and care of these patients. In some cases of sciatica the patients are robust and full-blooded and do not seem to require an extra full diet. The practitioner should not be deceived, however, by these appearances, especially if the individual be a man or woman who has been accustomed to high living with over-indulgence in alcohol. Such patients usually require a well-selected sustaining diet, but it should be simple and not disposed to aggravate a gouty diathesis. Not too much meat but light custards, milk, fish, and poultry, and a rather liberal vegetable dietary are indicated. In persons who are anæmic a full diet of easily digested food should be provided. Such patients often will not consume of their own accord as large an amount of nutritious food as they require, and their eating should in a sense be directed and even forced. With the treatment by rest, bandaging, and a splint, it is easy to control the patient's diet; and this is an additional advantage of this method of treatment. I think special attention to this subject of diet and nutrition is of great importance, particularly in cases of chronic sciatica. These patients are often broken down with pain, anxiety, and disappointment, and their cases become complicated with distinct neurasthenic symptoms. They consequently are benefited by rest, by forced feeding, and in some cases by removal from the domestic environment. I have known some of these patients to be at once benefited by removal from home to a

good hospital, where they could be kept thoroughly under control and systematically watched and treated.

The use of alcoholic stimulants in sciatica is, as a rule, not indicated. Old or broken-down subjects are, however, sometimes benefited by light wines or even a little whiskey.

Great care should be exercised in getting the patient up and about after treatment in bed for sciatica. If too much liberty is allowed at the beginning, there may be relapses with all their attendant discouragement and demoralization. It is best to allow the patient at first only to sit up in the chair for an hour or two in a day. If the improvement continues he may be permitted to walk a little about his room, care being taken to note the degree of pain, if any, that accompanies or follows the exercise. Slight soreness of the nerve trunk and a sense of stiffness of the muscles may persist even in favorable cases for quite a long time, and, while suggesting the necessity for caution, should not cause either the patient or the physician too much disappointment and anxiety. Even after the patient has apparently entirely recovered his health, he should live for months with a view to the possibility of a recurring attack. Consequently he should avoid all excesses and exposure in living and in labor. As a rule, even in very severe cases of sciatica I have not noted a tendency to recurrence after an interval of complete freedom of a year or two has passed by. In other words, recoveries from this disease are often both complete and satisfactory.

A word may be said here about the advisability of radical surgical treatment for sciatica. By this I mean cutting down upon the nerve and incising its sheath longitudinally for the purpose of securing drainage. In the case already referred to in this paper, in which I had an opportunity to make an autopsy in a patient who had been suffering with a high grade of sciatic neuritis, I was much impressed with the fact that only some radical treatment, like incision of the nerve sheath, would have been able to afford satisfactory and prompt relief. In this case the tough, unyielding fibrous sheath of the nerve contained lymph, blood, and serum, as the results of an acute inflammatory process. Pus even has been described by some observers as present in the nerve sheath. When such conditions exist in other regions of the body, the only rational treatment is deemed to be evacuation of these products. I do not know why a similar rule does not apply to the large sciatic nerve. Still, I have had no opportunity to test such treatment, and, therefore, I cannot with certainty recommend it. I should, however, in another similar case under my care certainly take the subject into consideration in consultation with a competent surgeon.

Diseases of the Peroneal Nerve.

The peroneal nerve is the continuation of the external popliteal nerve, which is one of the two main terminal branches of the great sciatic. It is a large nerve, but is nevertheless only about one-half the size of the internal popliteal. In passing into the leg from the popliteal space it lies behind the head of the fibula, where it can readily be felt. It divides into two terminal branches, the anterior tibial and the musculocutaneous nerve. Before its division, however, it supplies articular branches to the knee-joint and cutaneous branches to the skin on the back and outer side of the leg as far as its middle third. One branch occasionally extends as far as the heel (Gray). The anterior tibial nerve supplies the tibialis anticus, extensor longus digitorum, extensor proprius hallicis, and extensor brevis digitorum, and sends sensory filaments to the adjacent sides of the great and second toe. The musculocutaneous branch of the peroneal nerve supplies the peroneus longus and peroneus brevis muscles, and sends sensory fibres to the lower part of the leg, the inner and the outer side of the ankle and foot, the inner side of the great toe, and the adjoining sides of the second, third, fourth, and fifth toes.

Obstructive lesions of the peroneal nerve cause paralysis of the muscles just enumerated. There is consequently a loss of power of extension of the foot upon the leg (dorsiflexion) and of extension of the first phalanges of the toe. These phalanges are consequently flexed by contracture of the interossei. There is foot drop in walking, and a condition of talipes equinus from paralysis of the tibialis anticus and contracture of the unopposed sural muscles. The area of anæsthesia is confined largely to the outer side of the lower leg and ankle, to the outer side of the dorsum of the foot, and to the toes; but it varies considerably in different cases.

The peroneal nerve is not often paralyzed alone. Because of its exposed position at the head of the fibula, it is readily conceivable that the nerve might be injured by blows, wounds, or pressure. Practically, however, such cases must be extremely rare. I have never met with an instance of such an injury, nor seen a report of such a case. The nerve itself is not usually subject to the injurious effects of cold, such as is seen in the facial nerve. While thus exempt, however, from some accidents, it is rather unusually prone to be involved in some disease processes. Thus in the neuritis which sometimes follows typhoid fever the peroneal, as well as the ulnar nerve, is very likely to be involved. Sir James Paget,[1] in 1876, called atten-

tion to this type of paralysis following typhoid fever. Tooth[179] and Charcot and Marie[180] described a somewhat similar type of muscular atrophy in which talipes equinovarus occurred. Ormerod[181] reported three cases of this type of muscular atrophy in one family, following measles. The affection invariably began in the legs below the knees; then after some months, or even years, involved the hands and forearms, and always affected the distal more than the proximal portions of the limb. Trophic lesions resembling chilblains were observed. Electrotonus was abolished below the knees, and partial reaction of degeneration was seen elsewhere. The knee jerks were not abolished. Ross[182] believes that the typhoid poison shows a special predilection for the peroneal and anterior tibial nerves. Autopsies made by Virchow, Oppenheimer, Friedreich, and others have disclosed in cases of this peroneal type an interstitial neuritis. I have reported a case[183] in which the symptoms of peripheral neuritis, well marked in the peroneal muscles, although not confined to them, followed a prolonged attack of typhoid fever. This peroneal type of muscular atrophy has been ascribed usually to a peripheral neuritis. There are some difficulties, however, in the way of accepting this view. In some cases the presence of fibrillation, the absence of anæsthesia, the preservation of the tendon reflexes, and the partial character of the electrical changes, are not altogether in favor of the view that the peripheral nerves are always or alone in fault. Still, the result of autopsies has proved undoubtedly that, in some of these cases at least, the lesion is a neuritis, especially well marked in the peroneal nerve, but not confined to it, as the ulnar and some other nerves are equally involved.

Inflammation of the peroneal nerve causes flaccid paralysis with degeneration in the affected muscles. The reactions of degeneration, as a rule, are promptly established.

The *treatment* is in accord with that already outlined in other portions of this paper for peripheral neuritis. Massage, electricity, and the internal administration of strychnine are the most approved remedies.

Diseases of the Internal Popliteal Nerve.

This nerve is the larger of the terminal branches of the great sciatic. It sends branches to the knee-joint, and farther in its course is called by anatomists the posterior tibial nerve. It supplies especially the flexor muscles of the foot and toes; also the integument of the sole of the foot and the outer part of the back of the leg, and possibly the skin of the toes. But this sensory distribution appears to vary. The muscles supplied by the internal popliteal nerve are

the gastrocnemius, plantaris, soleus, popliteus, tibialias posticus, flexors of the toes, and the various muscles of the sole of the foot. An obstructive lesion of the nerve consequently causes great disability in the movements of the leg and foot. The foot cannot be flexed* at the ankle, and talipes calcaneus results. Anæsthesia is distributed especially on the outer and lower part of the back of the leg and on the sole of the foot. This nerve is seldom if ever paralyzed alone. Wounds and other injuries to it must be extremely rare, as I have been able to find no reference to instances of them.

The *treatment* is the same as that indicated for paralysis of the external popliteal nerve.

The Fourth and Fifth Sacral and the Coccygeal Nerves.

These nerves, the last in the spinal series, do not enter into the formation of the sacral plexus, except the fourth sacral, which sends a small branch to that plexus. The fourth sacral nerve is the most important of these nerves, since it supplies the levator ani, coccygeus, and sphincter ani muscles. It also sends branches to the viscera within the pelvis, and communicates with the sympathetic system, entering into the formation of the hypogastric plexus. It supplies sensory filaments to the skin back of the anus. The fifth sacral nerve supplies the skin over and to the side of the coccyx and supplies the coccygeus muscle. The coccygeal nerve exists sometimes in only a rudimentary state. It is distributed to the skin and fibrous tissue about the coccyx.

These nerves are seldom involved in a distinct peripheral lesion. They may be, and very commonly are, injured in lesions of the cauda equina. In the cases of injury to the sacral plexus by tumors or inflammatory exudates within the pelvis, these nerves are not commonly involved. This is true especially of the puerperal cases, in which the chief symptoms occur in the course of the great sciatic nerve. In such cases paralysis of the muscles of the anus is not commonly seen. Rare cases may occur in which a limited lesion at the very lowest portion of the spinal cord, *i.e.*, involving the last two sacral and the coccygeal segments, may closely simulate peripheral disease of these nerves. Thus Starr[187] relates the case of a young woman who was suddenly seized, after overexertion, with pain in the sacral region and down the back of the legs, with retention of urine and fæces, and with total anæsthesia of the vulva, vagina,

*I use the terms *flexion* and *extension* of the foot in their strictly physiological sense. The various muscles of the group supplied by the internal popliteal nerve are properly flexors, and are analogous to the flexors in the forearm.

and perineum, and of the limited ovoid area on the inner side of the buttock, which is innervated from the fourth and fifth sacral segments. There were no true paralyses, no atrophy of muscles, no reactions of degeneration, no electrical changes. In this case the very distinctly localized and outlined area of anæsthesia and the involvement of the bladder and rectum without paralysis of any other muscles, showed conclusively that the lesion must have involved either the last two sacral and coccygeal nerves, or their roots within the cauda equina, or their points of origin within the spinal cord. The fact that the lesion had come on suddenly, and especially that it was bilateral and symmetrical, and the absence of any recognizable lesion within the pelvis, pointed clearly to a disease of the spinal-cord segments rather than of the roots within the cauda equina or the nerves within the pelvis. The sudden onset of the symptoms in a young person after overexertion indicated, but did not prove, the presence of a spinal hemorrhage limited to the cord at and below the fourth sacral segment.

Lesions of these nerves within the pelvis would likely be unilateral, or if bilateral they would not present this appearance from the beginning—in other words, the symptoms would probably begin on one side and pass later to the other. The symptoms of involvement of the last two sacral and coccygeal nerves are paralysis of the bladder and rectum, and anæsthesia over the posterior part of the sacrum, the anus, perineum, and, in women, the vulva and vagina. In the paralysis of the bladder which results the detrusive power is abolished and the use of the catheter is demanded. If the bladder becomes greatly distended it may overflow, and a constant dribbling of urine may result, which may deceive an inexperienced observer into the belief that the bladder is not paralyzed in its detrusive but in its retaining power. Paralysis of the sphincter ani muscle leads eventually to an involuntary escape of fæces. In the early stages of these cases, however, there may be obstinate retention of fæces, requiring the use of enemata.

The *treatment* of these cases, as of all cases in which the bladder and rectum are paralyzed, requires great care, especially with regard to these organs. The use of the catheter, in all forms of nervous disease entailing paralysis of the bladder, carries with it a distinct peril to the patient. This peril arises from the possibility of septic infection, causing purulent cystitis with all its attendant evils. I have seen several cases, in my wards at the Philadelphia Hospital, of lesions of the spinal cord, causing paralysis of the bladder, in which the death of the patient could be directly and distinctly traced to the use of this instrument. These accidents happen in spite of the utmost care on the part of physicians and nurses to observe a rigid asepsis.

It is evident that in these cases, for some reason, the resistant power of the bladder is impaired, probably because its nutritive or trophic functions are interfered with. Consequently the irritation of the catheter is much more likely to be followed by a chill and the evidence of septic infection in these patients than in healthy men and women. It is astonishing in some instances with what promptness and rapidity the patient will succumb to the ravages of this comparatively harmless-looking instrument. I recently saw a young man with constitutional lead poisoning, which had induced partial paralysis of the arms and legs, complete secondary optic atrophy, and a marked lead dyscrasia, present symptoms of septicæmia within a few days after the commencement of catheterization. This patient died with all the symptoms of septic poisoning, and at the autopsy an extensive nephritic abscess was found, which had evidently resulted from a primary infection of the bladder. With these facts in mind, it seems that the practitioner must always view with concern the need of the catheter in paralysis of the bladder, and must realize the necessity for rigid asepsis and care in its use. Although, as already said, cases of paralysis of this organ from localized disease of the last two sacral nerves in the pelvis are rare, yet this seems an appropriate place to emphasize the dangers that arise from catheterization in all cases of paralysis of the bladder.

Diseases of the Cauda Equina.

The cauda equina is the leash of nerve roots lying within the spinal canal and extending from the lumbar and sacral segments of the spinal cord to the foramina by which the respective nerves make their exit from the spine. In other words, the cauda equina is composed of the roots of the lumbar, sacral, and coccygeal nerves. Some of these roots are very long, since they extend from the lumbar or sacral segments of the cord to their respective foramina low down in the spinal canal. As they are composed of the axis cylinders of the peripheral motor and sensory neurons, they belong properly to the peripheral nervous system, and therefore their diseases will be described here briefly. In one important respect, however, they have some characteristics of the central structures of the nervous system, *i.e.*, they are enveloped in the meninges. Consequently they are subject to some diseases, especially meningitis and the neoplasms which grow in the meninges, that properly belong to the central nervous system.

The nerve roots composing the cauda equina may be involved in numerous disease processes and in a variety of accidents. Meningitis

of syphilitic origin, although not so common in this region as higher in the cord, nevertheless occasionally implicates the cauda equina. Intraspinal tumors also occur in the cauda equina. Thus W. W. Fisher[184] has reported a case of a lobulated tumor growing from the pia mater at the lower end of the spinal canal, which was surrounded by the nerves of the cauda equina. Gowers[185] has reported a case of myolipoma attached to the conus medullaris, in which the nerve roots of the cauda equina were embedded. Hemorrhages within the spinal membranes may also involve this leash of nerves. Such hemorrhages are not infrequently caused by accidents, such as a fall from a height, in which the patient strikes upon the buttocks. Some of these cases are associated with fracture of the vertebræ, but this is not necessarily so in every case. Purulent infections of the lower end of the spinal canal occasionally occur. In one such case under my own care the infection apparently arose directly from an immense sloughing bedsore over the sacrum. The patient had been bedridden from a non-purulent myelitis, high in the dorsal cord. This lesion had no direct connection with the purulent infection of the cauda equina, except that it had induced the bedsore, which had caused in turn the infection of the lower end of the cord.

A brief consideration in detail of some points in the anatomy of the cauda equina and lower end of the spine, is essential to a right understanding of the lesions of this portion of the nervous system. These lesions have been studied with care and intelligence only in recent years, and we have even yet not accumulated sufficient data to permit us to draw positive conclusions in all cases. The most important of these cases for diagnosis and treatment are those that result from accidents. These are not very uncommon. In our large clinical service at the Philadelphia Hospital I have seen during the last ten years a comparatively large number of cases of injuries to the cauda equina. They are, however, sufficiently rare in general practice to cause them to be perplexing and troublesome cases to the physician. In the first place, it is well to recall that the lower end of the spinal column is the most massive portion of that structure, and that it is protected by large muscles. Consequently it is the part least liable to fractures. The only injuries that are likely to cause fractures of these bones are crushing and compressing accidents. Thus the fall of heavy weights, as masses of earth, coal, etc., may cause such fractures. Compression between the bumpers of cars, or, as I knew in one instance, between a canal boat and the side of a lock, may also fracture the vertebræ of this region of the spine. But it is not necessary that fracture of the vertebræ should be present in order to constitute a very grave lesion. Rupture of blood-vessels and slight

lacerations of the membranes may occur, without fractures, as results of violent concussion. These lesions are rather more common in the lumbar and sacral regions than are fractures of the vertebræ. The accidents that are peculiarly liable to cause them are falls from great heights, especially, I have thought, when the patients are heavy individuals and when they light upon the feet or upon the buttocks.

There seems to be some special reason why these meningeal hemorrhages affect particularly the region of the lumbar enlargement and the cauda equina. It is possibly due, in part at least, to two causes. In the first place, the direct effect of concussion, when the patient lights upon the feet or buttocks, is felt most in the lower dorsal and lumbar regions. Secondly, the blood-vessels are probably not so well supported in this lower region, since they have not the solid support of the spinal cord. There is also possibly a third reason: the blood, even when it escapes above the upper level of the cauda equina, would tend by gravity to run down within the membranes and to cause clots to form, which would make permanent compression upon the nerve roots. This seems to be indicated not only theoretically, but also from the clinical facts as they are observed. Thus the permanent symptoms of anæsthesia and paralysis do not always appear instantly at the time of or just after the accident. Many hours sometimes elapse before the symptoms of compression are permanently established. In one case reported by Mills,[186] in a man aged forty-four—who while carrying a hod fell from a scaffold twenty-five feet to the ground—fifteen hours elapsed before normal sensation and motion were abolished. Such a case can be accounted for only upon the hypothesis that some slowly acting cause with cumulative effects was taking place. This could only be a hemorrhage. This might not necessarily be a very copious flow of blood, but only a slight oozing or trickling from a few small blood-vessels. In the course of a few hours the blood could so fill up the lower end of the spinal canal as to cause very destructive pressure on the delicate nerve roots constituting the cauda equina. In some of these cases, no doubt, there is injury also to some of the segments of the lumbar and sacral cord. The difficulty often is to distinguish these lesions apart. An injury to a lumbar or sacral segment will necessarily cause symptoms almost identical with those caused by injuries to the nerve roots arising from these segments. It is in this one respect, perhaps, that we are still at a disadvantage in not having accumulated more data in these cases.

A second point of importance is to estimate the location or distribution of the lesion with reference to the exit of the various nerves

through their respective foramina from the spinal canal. The cauda equina, it must be recalled, is about six inches in length, estimating from the extreme tip of the spinal cord (the conus medullaris) to the exit of the last coccygeal nerve. Its component roots, however, rise, of course, above this level, and they are necessarily not all of the same length. Each pair of lumbar and sacral nerve roots is as long as the distance from their respective segments to their foramina of exit. Consequently the lower roots have the longer course. The important point to recall in this connection is the fact that these roots have different levels of exit, and consequently some may be involved in a lesion from which others may escape entirely. Thus a blood clot settling and organizing low in the spinal canal might only make compression upon the last two sacral nerve roots as they descend and emerge low down in the canal, while the upper sacral and all the lumbar nerve roots would escape, owing to the fact that they emerge above the seat of the lesion. This fact, it may be said, dominates, as it were, the topography of lesions of the cauda equina. The lower these lesions the more contracted is the field of their symptoms. With each additional level involved an additional pair of nerve roots is implicated, and the field of the symptoms consequently broadens out more and more the higher the lesion ascends. This practically amounts to the same thing as including within each additional level additional segments of the sacral and dorsal cord. From this fact arises the difficulty, already referred to, of distinguishing accurately between lesions purely of the cauda equina and those that are limited strictly to the cord itself. From the tables already given (page 256) it will be seen what sensory and motor neurons are connected with the various segments of the lumbar and sacral regions of the cord, and consequently what sensory and motor functions must be paralyzed in lesions involving the various segments. It is possible, in fact, or even probable that in many cases of meningeal hemorrhage both the segments of the cord and some of the various strands of the cauda equina are conjointly involved. In their efforts at refinement in diagnosis clinicians may perhaps seek to distinguish too dogmatically between lesions of these two structures, *i.e.*, the lumbar enlargement and the cauda equina.

The *symptoms* of diseases or injuries of the cauda equina present usually what may be called a distinctly segmental type; that is to say, they appear in groups in well-defined areas. The lower the lesion, the smaller and more contracted is the area of the symptoms. The higher we ascend, as segment after segment is added, the area of the symptoms, as it were, broadens out. Thus, beginning at the lowest segment, we can theoretically superimpose one segment

upon another, and represent diagrammatically each of the gradually increasing areas of the symptoms. In a lesion, for instance, of the fourth and fifth sacral segments or of the nerve roots running from them in the cauda equina, the area of anæsthesia is as shown diagrammatically in Fig. 35. This area includes also the perineum and the genital organs. With this anæsthesia there is always associated more or less paralysis of the bladder and rectum.

When the lesion involves the third sacral segment or its nerve

FIG. 35.—Area of Anæsthesia from a Lesion of the Fourth and Fifth Sacral Segments. (Starr.)

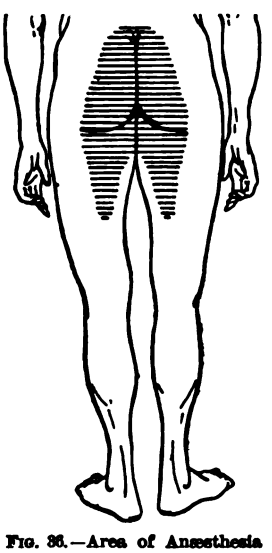

FIG. 36.—Area of Anæsthesia from a Lesion of and below the Third Sacral Segment, or the Nerve Roots below this Segment. (Starr.)

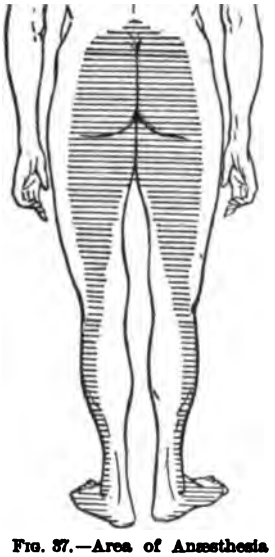

FIG. 37.—Area of Anæsthesia from a Lesion of and below the Fifth Lumbar Segment, or of the Nerve Roots from and below this Level. (Starr.)

roots, as well as those below it, the area of anæsthesia broadens out, as shown in Fig. 36. With this is associated, in addition to the paralyses of the rectum and bladder, paralysis of the gluteus maximus muscle.

When the lesion involves the fifth lumbar segment or its nerve roots, as well as those below it, the anæsthesia is as shown in Fig. 37. At the same time, in addition to the paralyses already mentioned, there will be loss of power in the outward rotators and flexors of the knee, flexors of the ankle, peronei, and extensors of the toes.

If the lesion extends as high as the third lumbar segment, the area of anæsthesia is as shown in Figs. 38 and 39. At the same time the paralysis will extend to the abductors and adductors of the thigh, the tibialis anticus, and the peroneus longus, but will not involve the quadriceps extensor (Mills, Starr).

This segmental distribution of the symptoms of lesions of the lumbar and sacral regions of the cord, or of their nerve roots in the cauda equina, is highly characteristic, and needs only care in the minute study of symptoms for its determination. In this study the area of anæsthesia can be mapped out very readily by means of a sharp and pointed instrument and a blue lead pencil. In determining the muscles involved it is only necessary to test each muscle separately and thus gradually establish the group paralyzed. By then com-

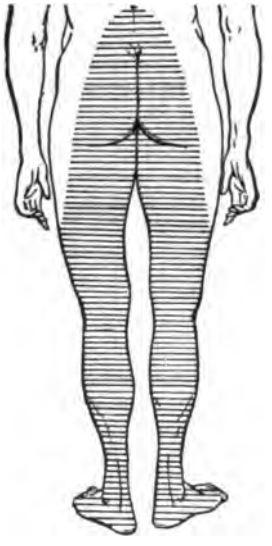

FIG. 38.—Area of Anæsthesia from a Lesion of and below the Third Lumbar Segment, or of the Nerve Roots at and below this Level. (Starr.)

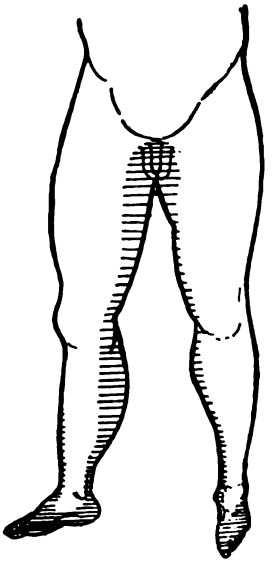

FIG. 39.—Same as Fig. 38, Anterior View. (Starr.)

paring the results with the tables given on page 249, a diagnosis of the seat and extent of the lesion can be established with great accuracy.

Among the general symptoms of all these lesions, pain is one of the most striking. This evidently depends upon irritation of the sensory nerve roots, and consequently may occur in a lesion involving these roots at any point of their course. Consequently it may be present in a lesion of the lumbar or sacral region of the cord itself or of the nerve roots distinctly limited to the cauda equina. The distinction which some authors have sought to make between these two classes of lesions, with respect to this one symptom, pain, is, I am sure, not a reliable one. In other words, there is no reason, as already

said, why pain should not be present in any irritative lesion, such as is produced by tearing of the meninges and pressure of a blood clot upon the nerve root anywhere in the spinal canal below the upper level of the lumbar enlargement. It has usually been present in some degree in all cases noted by me. According to Thorburn,[167] hyperæsthesia as a symptom is much less commonly found than the text-books indicate. This is probably due to the fact that his cases were mostly those of fracture in which a crush and compression of the cord itself had occurred. In such a case, the sensory pathways upwards in the cord being interrupted, pain would not be a prominent symptom. Hence this symptom, pain, in lesions in the lower dorsal, lumbar, and sacral regions of the spine is of relative diagnostic importance, as indicating that the nerve roots are chiefly involved and that the substance of the cord itself has not been seriously damaged.

The reflexes in lesions of the cauda equina will be affected according as their centres are located in the affected segments, the nerve roots of which are involved. These can be studied by reference to the table. The patellar tendon reflexes are usually not involved in any lesion below the third lumbar segment. It may occasionally happen, in fact, that from the extension upwards of the irritative process the knee jerks may be exaggerated. The genital reflexes are usually entirely lost. Neither priapism nor normal erection occurs.

As time advances the skin may assume a glossy hue, as seen in cases of neuritis, and trophic lesions may occur. Bedsores not uncommonly form and seriously complicate the case. Paralysis of the bladder may lead to cystitis and ammoniacal urine.

The paralysis in these cases is of the flaccid type. The affected muscles degenerate and present the reactions of degeneration. This, of course, is due to the fact that the lesion is essentially peripheral, *i.e.*, it involves the axis cylinders of the peripheral motor neurons. Consequently the muscles are cut off from their trophic centres, the large multipolar ganglion cells in the anterior horns of the spinal cord. The lesion acts, therefore, exactly as a lesion would in the course of a nerve trunk.

The peculiar distribution or grouping of the symptoms of hemorrhage in the cauda equina is well illustrated in a case reported by Mills[168] from the neurological wards in the Philadelphia Hospital. The patient, a man aged forty-four, born in Ireland, a laborer, fell a distance of twenty-five feet and lighted on his feet. From the beginning he could not walk. There was no fracture of the vertebræ. For the first fifteen hours he had normal sensation in his legs, and he could move his legs up and down as he lay in bed. He had severe

pain in his back, in the pelvis, and in the gluteal region. At the end of fifteen hours pains and cramps in the left foot and leg set in, and the leg became swollen and blue. These symptoms then appeared in the right leg and both legs were drawn up, *i.e.*, flexed at the hip from contraction of the quadriceps extensor muscle. The legs were so paralyzed that they had to be lifted about when it was desired to change the patient's position. Incontinence of urine set in at the end of fifteen hours, and constipation of the bowels was a symptom for six months, after which occasional incontinence of fæces occurred. Paræsthetic symptoms continued for a long while in this case, but pain was not a very prominent symptom. The paræsthesia consisted of a tingling in the calves and posterior part of the thighs and buttocks, and in the left leg of a sensation of a stream of water flowing down the inner side of the thigh and leg. The knee jerks were exaggerated. In this case the anæsthesia was in the distribution of the small sciatic, pudic, inferior hemorrhoidal, and inferior pudendal nerves. A surrounding and larger zone of partial anæsthesia was observed. The case thus illustrates the important point, that a larger zone of imperfect anæsthesia may surround the smaller zone. This indicates that the segment or the pair of nerve roots just above those most injured were also partially involved. This fact, which is an important one, has been in my observation too generally overlooked by clinicians. Analyzing the motor symptoms, we find that the affected muscles were mostly those below the knee, as the gastrocnemius, soleus, posterior tibial, and, in a minor degree, the anterior tibial. This distribution of symptoms indicates that the lesion involved mostly the lower sacral segments, but that it extended upwards with irregular or partially destructive effects as high possibly as the fourth or even the fifth lumbar segment.

The roots of the cauda equina may be involved, as already stated, in acute inflammatory processes not the results of accident. Syphilis and septic processes may cause an inflammation of the membranes involving the roots of the cauda equina. Such patients usually suffer extreme pain. I have seen a case presenting distinct symptoms of inflammation of some of the lower strands of the cauda equina as a result of exposure to cold. The case was as follows:

J. B——, an able-bodied Italian, aged 40 years, was admitted to my ward in the Philadelphia Hospital, with well-marked paralysis of the lower limbs. Four months before his admission he had been working in midwinter in a railroad tunnel, where he had been constantly exposed to cold and wet. He had no history of alcoholism or syphilis. The first symptom noted had been shooting pains down the legs; at the same time he began to lose power in his legs, the

paralysis advancing rapidly. On admission the patient had paralysis of certain groups of muscles of the legs and thighs. Those most affected were the sural and peroneal groups and some of the thigh muscles, especially the hamstring muscles. There was not complete paralysis, however, as the patient was able to flex and extend his thighs and to stand with support and to walk with a very feeble gait. He had the characteristic gait that is caused by paralysis of the extensor muscles of the foot; he dragged his toes and lifted his feet very high. In other words, there was foot drop. The symptoms in the legs, in fact, closely resembled those of a patient suffering with multiple neuritis. He had no symptoms, however, above the waist, and the absence of multiple neuritis was further indicated by his sensory and vesicoanal symptoms. The paralyzed muscles were atrophied and the knee jerks were abolished. There were no contractures. The patient swayed slightly with his eyes shut. The faradic contractility was entirely abolished in all the muscles of the leg and the posterior thigh muscles. The quadriceps extensor group responded sluggishly to strong currents. To galvanism there was much diminished contractility in the affected muscles, with decided modal change, and a duration tetany to very strong currents was obtained. Serial changes were well marked. The CCC was less than the ACC in all the muscles below the knees. In the thigh muscles the CCC was equal to the ACC. It is thus seen that the patient had complete reactions of degeneration. Sensation was affected as shown in the diagrams. The anæsthesia involved the buttocks and extended in a strip down the posterior part of the thighs and legs, passing around to the outer anterior aspect slightly on the thighs but farther on the leg and including the dorsum of the foot. All modes of sensation were involved, *i.e.*, tactile, thermal, and the pain sense. The patient at this time had no pain whatever. The bladder was paretic but not totally paralyzed. The man had difficulty in starting the stream, but no incontinence. The rectum at this time was not involved. There was a bedsore on the right buttock. In the course of two months the patient's condition improved. The area of anæsthesia diminished, the right knee jerk returned, and there was some gain in power. Later, however, a second bedsore appeared. These bedsores had a punched-out appearance; they were painless and exceedingly sluggish. The gait improved steadily up to a certain point, but the foot drop continued and was a troublesome feature. The patient had a device for overcoming the foot drop, by tying a bandage about the ball of the foot and passing one end to the outer

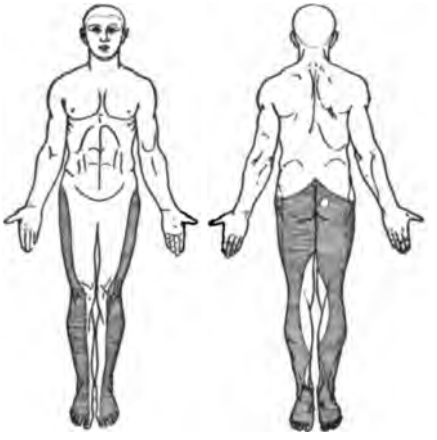

FIG. 40.—Area of Anæsthesia in a case of Neuritis of the Cauda Equina. (Philadelphia Hospital.)

and the other to the inner side above the calf of the leg, where they were fastened.

Analysis of this man's symptoms shows that the lesion probably extended as high as the fourth lumbar segment. From the mode of its onset and its obvious cause, it was probably an inflammation of some of the lower nerve roots of the cauda equina. In the absence of a distinct history of traumatism, syphilis, and alcoholism, and from the direct history of exposure at hard labor in a cold and wet place, it seems obvious that it was a neuritis due to the action of the cold and wet.

The *diagnosis* of lesions of the cauda equina rests largely upon the peculiar segmental distribution of the symptoms. The nature of the lesion is usually clearly indicated by the history of the case. The majority of these cases are of traumatic origin. In all of them the symptoms are usually bilateral and symmetrical. Exceptions to this rule might occur possibly in cases of tumor compressing the cauda equina, in which cases the first symptoms at least would probably be unilateral. In most cases of lesion of the cauda equina pain is a prominent symptom at some stage in the history. It may be well marked, however, only in the early stages. In the case just narrated it disappeared after the lapse of a few weeks. In a case of purulent infection of the cauda equina, seen by me, it was a well-marked and persistent symptom to the very end. The diseases from which these lesions have to be differentiated are especially myelitis, locomotor ataxia, and peripheral neuritis. These lesions of the cauda equina are best differentiated from all these diseases by the peculiar segmental grouping of the symptoms. In myelitis involving only the lumbar and sacral segments, however, this differentiation is not always easily made. In one of my cases in the Philadelphia Hospital the patient presented quite distinct segmental symptoms, and the autopsy revealed a diffused myelitis, with white softening, extending from the lower dorsal segments to the filum terminale. It involved some strands of the cauda equina. There were, however, no reactions of degeneration, and the presence of fibrillation in the muscles with exaggerated knee-jerks and the absence of pain admitted of a diagnosis (which was made before death) of irregularly diffused myelitis of the lumbar cord. From locomotor ataxia the disease is to be distinguished especially by the history and by the fact that in tabes there is no distinct segmental anæsthesia; that the fulgurant pains differ in type from those of neuritis; that the muscles, at least in the early stages, are not paralyzed and degenerated; that incoordination is well marked, and that eye symptoms are usually present. Multiple neuritis does not involve the legs alone, and the

paralysis of the leg muscles in this disease is not confined so distinctly to certain segmental groups as in the cases of lesions of the cauda equina.

The *treatment* of these cases depends largely, of course, upon the cause. In traumatic cases, in which there is good reason to believe that hemorrhage has occurred and that a blood clot is pressing upon the nerve roots, the only rational treatment is to trephine and evacuate the blood. This is often a difficult question to determine. Much depends upon an exact diagnosis of the lesion and its location. If good is to be obtained, the operation should be performed as soon as possible after the accident. In saying this much, however, I do not intend to recommend rash and hasty operative interference in these cases. The question can only be decided on its merits in each individual case in consultation with a conservative surgeon. The treatment of these cases with drugs is usually most unsatisfactory. In spite of time and rest and careful nursing, the patients are only too likely to be permanently impaired. Not a few of them, in fact, perish ultimately from exhaustion and from septicæmia due to bedsores. In later stages in cases in which the patients show a tendency to improve, massage and electricity with appropriate nerve tonics may be used. Bedsores should be treated with the utmost care and with due regard to asepsis.

MULTIPLE NEURITIS.

Definition.

Multiple neuritis, or polyneuritis, or peripheral neuritis, is an inflammation of a few or many nerves more or less limited to their peripheral distribution. Before entering into a description of this disease it is desirable to explain and limit the term so as to bring it within the meaning now generally accepted by neuropathologists. All inflammatory, and especially all degenerative, processes involving the peripheral neurons are not necessarily included in this term. As Babinski [188] has well pointed out, degenerative and inflammatory processes of nerves may be divided into two classes. The first of these pertain to those processes which depend upon lesions of the nerve centres themselves. Thus an inflammation of the anterior horns of the spinal cord, in which are contained the cell bodies of the peripheral motor neurons, will cause parenchymatous degeneration in the nerve trunks; but such a disease is not properly included with instances of multiple neuritis. The second class includes instances of neuritis properly so called which are peripheral and primary. In

these cases the nerves are attacked primarily in their trunks or distributions, and not secondarily to an inflammatory process in their trophic centres. This inflammation is more marked in the periphery of the nerves and tends to diminish towards the nerve centres, which, as a rule, are little if at all inflamed. It is proper to state here, however, that even in this class of true peripheral neuritis authorities differ as to the exemption of the trophic centres. This class may be subdivided, therefore, into first, those cases which have an external cause, as trauma, compression, etc., and which are undoubtedly truly peripheral in origin and course; and second, into an exceedingly large class in which the cause is internal and is usually a poison of some sort circulating in the blood. In these cases—which are the cases proper of polyneuritis—some authorities claim that the morbid process, though most marked in the periphery of the nerve, is nevertheless not limited to it or even primary; but that the trophic centres of the neurons (*i.e.*, the cell bodies proper) are also invaded by the poison which, in fact, acts primarily upon them.

Leaving aside for the moment these complex questions in pathology, the term multiple neuritis may be limited to that disease process which is set up in the trunks and distribution of a number of nerves at the same time, and is caused by the irritant action of some toxic substance circulating in the blood. As a consequence of this origin the disease is usually symmetrically distributed, although its exact distribution depends in a measure upon the particular poison in action.

A second question of importance has reference to the particular structure of the nerve trunk most involved in the inflammatory process. Two classes are here recognizable. First, those cases in which the nerve sheath and interstitial connective tissue are primarily and most involved. Second, those cases in which the inflammatory process is located especially in the axis cylinders. The first form is called interstitial neuritis; the second, parenchymatous neuritis. The distinction between these two varieties is a purely pathological question and will be discussed later under its appropriate head.

History.

The history of the recognition of multiple neuritis is one of the most important chapters in modern neuropathology. It seems almost incredible that such an easily recognized and prevalent disease should for so long a time have escaped recognition by clinicians. The reason for this, however, is not far to seek. This disease was uniformly confused with diseases of the spinal cord. These

cases were not unobserved but badly observed, and their morbid anatomy was not recognized because the science of pathology had not advanced far enough to admit of a recognition of diseased structures which are now open to the scrutiny of all experts with the microscope. As is so often the case, however, the pioneer in this field had few followers. The disease was distinctly recognized and described from the clinical standpoint by an American physician, Dr. James Jackson,[189] in 1822. His description is a model of conciseness and accuracy. He says that the disease arises from the use of ardent spirits; that it is distinctly marked but that he had never seen it described. He had seen a number of cases mostly among women, and for want of a better name he proposed to call the disease *arthrodynia a potu*. Jackson said that the disease commences with pains in the lower limbs, especially in the feet, and afterwards extends to the hands and arms. He called attention to the excruciating pain and the accompanying distressing feeling of numbness; also to the fact that the extensor muscles are most involved and that the flexor muscles, manifesting greater power, cause contractures. He spoke of the atrophy of the limbs, and of the altered appearance of the skin, which consists "in a great smoothness and shining with a sort of fineness of the skin." His description, in fact, of glossy skin is striking. The integuments, he says, look as if tight and stretched without rugæ or wrinkles, somewhat as when the adjacent parts are swollen. Yet this appearance he says is not due to an effusion under the skin; it arises from some change in the integument itself. He distinctly claims that the paralysis is due to some affection of the muscles and not of the nerves (*i.e.*, nerve centres ?) as in common paralytic cases. He thus nearly recognized very shrewdly the commonest seat of the disease, the peripheral distribution of the nerves in the muscular tissue; but he gave no anatomical demonstration of this. Finally, he recognized the altered cerebral state, the mental weakness and the insomnia. The disease, in his observation, was fatal if the use of spirituous liquors was not abandoned. In one case he effected a cure after six months' treatment, illustrating what is now so well known, the slow and chronic course of the affection.

Soon after Jackson's description an epidemic, apparently of some form of multiple neuritis, was observed in Paris and described by Chomel, quoted by Buzzard.[190] This was in 1828. Graves refers to this as a remarkable example of disease of the nervous system, commencing in the extremities, "and having no connection with lesions of the brain or spinal marrow." Graves saw some of these cases, and states that the disease began with pricking sensations and severe pain in the hands and feet, so acute that the patient could not bear these

parts to be touched by the bedclothes. Finally, anæsthesia came on and motor paralysis. All four extremities were involved, so that the person lay in bed powerless and helpless. The disease continued for weeks and even months. According to Graves, "the French pathologists searched anxiously in the nervous centres for the cause of this strange disorder, but could find none. There was no evident lesion, functional or organic, discoverable in the brain, cerebellum, or spinal marrow."

Huss, in 1852, and Duchenne[1] in 1855 undoubtedly observed and described cases of multiple neuritis, but without recognizing the true nature of the disorder. The former writer, according to Starr,[2] gave a description of the nervous symptoms in chronic alcoholism, but ascribed them to lesions in the central nervous system. Duchenne included these cases distinctly in a spinal group under the term of "ascending subacute general spinal paralysis," in spite of the fact that in a case in which he made an autopsy there were no gross changes observable in the cord. It thus appears that neither Huss nor Duchenne can receive credit for having thrown light upon the true pathology of multiple neuritis. They, in fact, did not advance so far even as the American physician, Jackson, thirty years before them, nor the Irish physician, Graves, in his observations while in Paris.

The first observer to demonstrate the true pathology of multiple neuritis was undoubtedly Duménil.[3] The title of Duménil's first paper sufficiently indicates his conception of this disease. It is as follows: "Paralysie périphérique du mouvement et du sentiment portant sur les quatre membres; atrophie des rameaux nerveux des parties paralysées." Duménil's first observation was made in the case of a man seventy-one years of age, who experienced first for a period of two weeks tingling sensations in the toes. These were followed by numbness in the left foot and in the right upper limb. Many days later the left upper limb was seized and finally the right foot was attacked in its turn. When the disease became established the symptoms were briefly as follows: There was paralysis, not quite complete, of the hand and of the forearm, especially on the right side; paresis of the left hand; almost complete paralysis upon the left side. There was atrophy of the muscles of the forearm and especially of those of the hand. Faradic contractility of the flexors on the right side was abolished and on the left side was diminished, and of the muscles of the thenar eminence and of the interossei it was abolished on both sides. There were areas of anæsthesia in the hand. The toes were completely paralyzed. The muscles of the legs were atrophied and their faradic contractility was abolished.

There was some anæsthesia on the dorsum and sole of the foot. The patient had a painful, numb feeling in the paralyzed limb. Sometimes the painful sensations were as though caused by the pricking of a needle. The heart beat irregularly. This patient died in two months after the onset of the malady of a latent pneumonia. A microscopical examination made by Duménil in this case showed the presence of pronounced changes in the nerves. The examination, however, was not so complete as it might have been, as the nerves were not examined from the periphery up to their centres. From gross appearances, however, it seemed that the spinal nerve roots and the cord itself were normal, but they were not examined microscopically. This autopsy, somewhat imperfect as it was, is noteworthy as being the first in which was demonstrated the disease now known as multiple neuritis. It may, therefore, justly be regarded as marking an epoch.

In his second contribution Duménil distinctly asserts his conviction that many paralyses of obscure cause have their origin in true neuritis. He ascribes the neglect of this subject to the preoccupation of pathologists with affections of the nerve centres, whenever they observed a trouble of sensibility or motion, and to the fact that this had distracted their attention from diseases of the peripheral nervous system. His second case in a man aged sixty-four was marked by sensory disturbances in the feet and hands; paralysis of the hands, of the forearms, and of the feet and legs; inability to extend the feet; abolition or enfeeblement of faradic contractility; cutaneous anæsthesia; pains in the feet and legs; swelling of the phalangeal articulations; fusiform swelling of the fingers, which were smooth and shining. These symptoms abated after some months, and the patient quit the hospital much improved. The diagnosis of multiple neuritis in this second case of Duménil's was amply justified by the symptoms, but as it was not verified by an autopsy, the case itself, of course, has not the importance of his first observation. Whatever criticism may be passed upon these observations of Duménil, they did not receive at the time nearly the attention that they deserved. The consequence was that the more general recognition of this important disease was postponed still for a number of years. For a more detailed statement of this interesting subject the reader may consult the article by Babinski.[100]

In 1876 Eichhorst, quoted by Babinski, reported a case under the title of acute progressive neuritis, which resembled Landry's disease. At the autopsy lesions of the nerves were found, while the spinal cord was normal. This observation has historic significance as being probably the first in which the identity, of some cases at least, of

so-called Landry's paralysis with multiple neuritis was demonstrated.

The next important paper on this subject was that by Joffroy[11] on spontaneous parenchymatous neuritis, which appeared in 1879. He distinguished three kinds of parenchymatous neuritis, and thus advanced the subject by recognizing and classifying a variety of causes. The first of these causes, according to Joffroy, was cold. He believed that the action of cold, especially when associated with a rheumatic diathesis, can develop neuralgias which are dependent upon parenchymatous neuritis. The most remarkable example of this, according to Joffroy, is sciatica. The second cause of parenchymatous neuritis was lead poisoning. In this observation, however, Joffroy had been preceded by Charcot and Gombault. The third cause, following this writer, was infection with the poisons of such diseases especially as typhoid fever, typhus fever, small-pox, and diphtheria. The merit of Joffroy's work, as already said, was the recognition of a variety of causes for multiple neuritis, but it is extraordinary that among these causes he made no mention of alcohol. He recognized, however, the action of tuberculosis in causing the disease, an action which was recognized the same year independently by Eisenlohr.

In 1879 and 1880 appeared Leyden's classical essays upon this subject. These papers advanced the subject and placed it upon a still more secure foundation. This observer devoted himself to a detailed description both of the symptoms and of the microscopical appearances. His observations were based upon two cases, both in young men in whom a paralysis of all four limbs had developed. This paralysis was especially marked in the fingers and hands and toes and feet. The paralyzed muscles atrophied and reactions of degeneration were present in them. Tormenting pains were experienced in the paralyzed limbs. Leyden noticed also the hyperæsthesia of the skin and the pain caused by compression of the muscular masses. He emphasized the fact that characteristic symptoms of spinal diseases were absent, especially paralysis of the bladder and rectum, bedsores, and involvement of the eye muscles (Babinski). Microscopical examination in Leyden's cases revealed characteristic alterations of the nerves without involvement of the spinal cord. The value of Leyden's observations consisted in the greater detail with which he gave the symptoms of the disease and the accuracy and fulness of his histological studies. While he merely followed in the path already so clearly indicated by Duménil and pursued by Joffroy, he has the merit, perhaps more than any other author, of having secured for the disease a recognized place in nosology.

Among British writers, special mention should be made of a paper by Grainger Stewart.[115] This paper is noteworthy as the first attempt in the English language at a systematic description of the disease as it is recognized by modern pathologists. The paper was based upon the report of two cases. The symptoms in these cases were in the main similar. Pain and numbness in the extremities, followed by anæsthesia and paralysis were the chief symptoms. Grainger Stewart also called attention to the intermittent character of the pain, which was increased on pressure and handling; also to the glossiness of the skin, and the contractures and changes in the electric excitability of the muscles. He did not, however, appear to recognize fully the possible alcoholic element in his cases. One of his patients died, and the microscope revealed marked changes in the median, ulnar, and tibial nerves. There was destruction of the axis cylinders, which, however, was only apparent in the trunk of the nerve and towards the periphery and not towards the centres. Thus the brachial plexus was exempt, while the nerve trunks arising from it, just mentioned, were the seats of disease. The only changes found in the spinal cord were in the columns of Goll and the posterior part of the lateral columns, in the cervical region and to a less extent in the lumbar enlargement. The roots of the nerves were not involved and the gray matter in the cord also was exempt.

It is scarcely worth while, or even possible, in this place to trace the history of multiple neuritis farther. The disease, after its full recognition, became the subject of innumerable papers which now constitute an enormous bibliography. The observations of clinicians and pathologists all over the civilized world have established the fact that there are numerous varieties of the disease and that they depend upon many causes.

In this paper it will be my plan to describe in detail the various symptoms according to the structures involved; then to group these symptoms in the various forms which multiple neuritis actually presents at the bedside. The diagnosis will then be considered in considerable detail. Next the morbid anatomy will be described, and finally, in the section on treatment, care will be taken to indicate the various methods by which the disease is to be controlled. First, however, I shall describe fully the numerous causes of multiple neuritis.

Etiology.

Not the least interesting phase of the study of multiple neuritis is its etiology. Since the disease was first demonstrated as a clinical entity, our conception of its possible causation has been constantly

widening until now we realize that the peripheral nerves are the field for the utmost activity of most of the poisons, both organic and inorganic, that can infect the human body. This fact shows us at once that we are now on a most important territory in neuropathology. Many diseases that were formerly considered to have their seat in the spinal cord have now been demonstrated to be simply due to the action of some poison upon the smaller nerve trunks and their peripheral endings. No other department in clinical medicine equals this in suggestiveness and in the fertility with which it has given origin to new conceptions of disease.

Among the causes of multiple neuritis *alcohol* should undoubtedly hold the first rank. As already said, Magnus Huss had noted the symptoms of peripheral neuritis in alcoholic subjects, but he had not attributed them to their true cause. Long before his time, however, Lettsom, quoted by Buzzard, had made a curious reference to the same subject. This was in 1789. He referred to the emaciation of the extremities; the smoothness and polish of the skin, so that the soles of the feet even became "glossy and shining"; and the exquisite tenderness of the parts, so that the "weight of the finger excites shrieks and moaning." He wrote especially of the paralysis of the legs, arms, and hands, of the mental feebleness, and of the delusions in particular of personal identity. In recent years the study of alcohol as a factor in neuritis has been made by Lancereaux and Leudet in France, and by Reginald Thompson, Handfield Jones and Wilkes[m] in England. This latter author, however, ascribed the "alcoholic paraplegia" to an infection of the spinal cord. He called particular attention to the fact that the disease occurred especially in women. He speaks of the loss of power, accompanied with pain in the limbs—in some cases, with anæsthesia—but he erroneously ascribes these symptoms to spinal meningitis. Wilkes gives interesting details of cases evidently of multiple neuritis in women. He notes the feebleness of mind as well as the impairment of the body and says he could multiply such cases to almost any extent. Alcohol acts to cause multiple neuritis often in an insidious manner, and in conjunction especially with one or two other well-marked causes. The disease usually appears, for instance, in the chronic tippler and not necessarily in one who has drunk large quantities of spirituous liquors. For this reason, probably, it is seen so often in women, many of whom, when addicted to drink, take it on the sly and in small quantities at brief intervals. When this habit of constant tippling is associated with hard labor, exposure to changes and extremes in temperature, and insufficient food, the chances are much increased for the occurrence of the disease. These

facts hold good, however, just as well for the male as for the female sex. Thus most of the severe cases of alcoholic multiple neuritis in men which I have seen in the Philadelphia Hospital, have occurred in the pauper class and in individuals who were especially exposed to cold, wet, and fatigue, and who not only drank too much, but ate too little. Thus one man was a blind street musician, who exposed himself on a curbstone for many hours a day and spent all his earnings in low groggeries. Another patient was a chronic tippler, much reduced by exposure and neglect, whose disease appeared after a few days' exposure shoveling snow in severe winter weather. One reason, apparently, why women are so prone to the disease is probably because those women in whom it occurs most frequently are among the poorer classes, who are obliged to labor hard for the support of a family and are broken down often by over-childbearing and underfeeding. It is a mistake, however, to suppose that multiple neuritis is limited to this class. It may occur among the affluent classes. If it occurs less frequently among them, it is not probably altogether because they drink less, but because they are better nourished and hence can resist better the ravages of the disease. Because of its poisonous action upon other organs and tissues of the body, the effects of alcohol are not always limited to the peripheral nerves. Mental enfeeblement, marked by wandering delirium, is a very common symptom, and is an evidence of the ravages of alcohol upon the brain substance. Affections of the liver, kidneys, and vascular system are also liable to occur, and should always be sought for. These complications not only tend to give some distinctive characterization to the cases of multiple neuritis caused by alcohol, but they also not infrequently influence the course and termination of the disease.

The next most common cause of multiple neuritis is probably *lead*. This cause is not always recognized at its full value. The conception of "lead palsy" is too closely limited to a paralysis of the extensor muscles of the forearm. This is, of course, a variety or type of multiple neuritis, because in it, although the disease affects little more than one nerve of the forearm, it is nevertheless bilateral and symmetrical. It is a mistake, however, to suppose that lead does not cause in some instances a more widespread destruction. This poison can, for instance, inflame especially the nerves supplying the deltoid and other muscles in the shoulder and upper-arm groups. In some cases, too, it invades the nerves of the legs, and when it is thus widespread it may cause a state that may have some resemblance to the multiple neuritis of alcoholic origin. In my observation, however, the multiple neuritis of lead is not apt to be so widely

and so uniformly distributed as is that caused by alcohol. This poison also occasionally produces a type of multiple neuritis which has been called "pseudotabes," in which the sensory nerve endings especially are involved and in which the predominant symptom is ataxia. This type will be described later, with report of a case. The peripheral neuritis caused by lead has been especially described by Gombault,[197] who found it most marked in certain nerve trunks, especially the radial (or posterior interosseus?), as is well known clinically, and he described it as being of the segmental type, *i.e.*, the inflammation is confined to some segments of the nerve. Gombault's observations (made in 1873) are especially noteworthy, as they were among the earliest to lead to a general recognition of the fact that lead is a cause of peripheral neuritis. He drew attention particularly to the symmetry of the lesions in the absence of involvement of the central nervous system. Multiple neuritis caused by lead is usually observed in persons who have been exposed to the poison for long periods and have probably absorbed it only in small doses at a time. Thus it is seen especially in artisans who work in lead, as, for instance, workmen in white-lead factories and painters. In these persons the poison is probably largely absorbed from the alimentary tract. Thus in white-lead factories, in which the air is charged with dust, the lead is probably mixed with the saliva and so carried into the stomach. Among painters and other workmen who are careless in their habits, it is probably taken in minute quantities with the food, which the workman holds with unwashed hands. Lead is used so extensively in the arts that it may manifest itself in numerous other instances. Some of the most common of these are in lead miners and smelters, in workmen who roll sheet lead, shot makers, type founders, typesetters, plumbers, lace makers, glass cutters, glaziers—who may absorb lead from putty, file cutters, and calico printers. For details of the effects of lead as encountered in the occupations, the reader may consult the author's paper on the Diseases of Occupations in Vol. III. of this system. The preference of lead for some nerves, especially the radial and posterior interosseous, is not readily explained. The poison seems to have a selective action for these particular nerves, just as atropine, morphine, and other organic poisons have for particular nerve structures. The reason for its more widespread effect in those particular instances in which it causes a more general multiple neuritis is also obscure. It produces a type of multiple neuritis which differs in several respects from that caused by alcohol. For instance, sensory symptoms, and especially pain, are not nearly so conspicuous.

Of great importance as exciting causes of multiple neuritis are the

poisons of the various *infectious diseases.* The first place among these should be given to *diphtheria.* It has long been known that a form of paralysis sometimes follows inflammatory diseases of the throat. One of the earliest observers who recorded such cases was Bard, of New York, who, in 1771, published an inquiry into the nature, cause, and cure of a disease which he called angina suffocativa or sore throat distemper, in which he noted the occurrence of paralysis. Earlier observations are said to have been made by Lepois in 1580 and by Ghisi, an Italian physician, in 1747. Chomel also has a reference to the disease having occurred in France in 1748. Bard's observations, however, seem to have been the most accurate and detailed. During the present century the occurrence of diphtheritic paralysis has been generally recognized, but the identity of the disease as a form of multiple neuritis dates from a comparatively recent period. This identity has been satisfactorily established not only by anatomical studies on the human subject, but also by experiments upon animals. Among the most important of the latter are those by Roux and Yersin, who inoculated animals with the toxins of diphtheria and caused a paralysis similar to that which occurs in man.

Sydney Martin, quoted by Osler,[1] extracted from the spleen and blood of persons who had died of diphtheria an albumose which produced in animals, when injected under the skin, a paralysis due to degeneration of the axis cylinders of the nerves. Other symptoms caused by this albumose were fever and emaciation. The inference is that the cause of diphtheritic paralysis is a ferment produced by a bacillus, and that this ferment is the poison which causes not only the paralysis, but the fever and wasting and other symptoms of infection seen in diphtheria. The diphtheritic poison acts slowly upon the nerves, as a rule, in the human subject, or, at least, is not liberated until convalescence from the primary disease is well established. Thus it is not uncommon for the patient to have entirely recovered from the local disease in the throat, and to have even got about, before the symptoms of involvement of the peripheral nerves manifest themselves. I knew one instance, in a child, in which the paralysis did not occur until the fifth week. The reason for this latency of the diphtheritic poison for such comparatively long periods is not plain.

Martin, in his observations just quoted, inclines to the belief that these toxins are stored in the spleen. His explanation is that the ferment digests the proteids of the body, forming albumose and an organic acid, but that as a relatively large proportion of these proteids stagnate in the spleen, the greatest quantity of the ferment

is located there. It may be, according to this view, that the explanation of the latency of the diphtheritic poison is to be found in this fact, *i.e.*, that the poison is stored up, as it were, in the spleen, and only liberated gradually. Some such explanation as this seems to be necessary for the peculiar action of the ferment. The old view, that the inflammation spread by contiguity directly from the inflamed tissues of the throat by way of the pharyngeal nerves into the nervous system, is not tenable. The poison usually acts upon various and widely distributed nerves, often simultaneously. It must therefore be carried into the blood, and in cases in which its action is postponed for days or even weeks after the primary infection has subsided, it must have been retained during this interval in some reservoir, as it were, within the body. Diphtheritic paralysis does not necessarily occur after every malignant attack of diphtheria, nor is every case the result of a malignant attack. In other words, there is no definite connection between the severity of the primary attack and the occurrence of the paralysis. Severe and long-continued symptoms sometimes follow mild attacks of sore throat. It has even been claimed that non-diphtheritic sore throat may be followed by paralysis, but this is undoubtedly an error. The data of bacteriology are now sufficiently secure to prove incontestably that the peculiar paralyses caused by infective sore throats are always due to a specific poison generated by the bacillus of diphtheria.

Typhoid fever is occasionally followed by or complicated with the symptoms of multiple neuritis. This complication or sequel has been recognized for some years, the recognition having been strictly contemporaneous with that universal recognition of multiple neuritis which, during the last two decades, has placed that affection among the most common of nervous diseases. I have discussed this subject in my paper on "Muscular Atrophy and Peripheral Nerve Changes following Typhoid Fever," already cited, and from which quotations are made here in the text. Typhoid fever probably comes next in order to diphtheria as the infectious disease that most commonly causes multiple neuritis. The various paralyses following enteric fever were observed, of course, long before their true nature was recognized, and were almost universally ascribed to involvement of the spinal cord. Among the earliest observers to recognize the occurrence of neuritis in typhoid fever was Sir James Paget,[178] who in 1876 wrote that the peroneal and ulnar nerves were especially liable to the invasion of this poison. About the same time Tooth, and then Charcot and Marie, described a peroneal type of progressive muscular atrophy. Murchison noted hyperæsthesia in the lower extremities in about five per cent. of his cases of typhoid fever; and Hutchinson[179] had seen this hyperæs-

thesia so marked that the lightest touch made the patient cry out with pain. This hyperæsthesia or hyperalgesia, especially in the legs, even on slight contact of the bedclothes, is well shown in cases recently reported by Preston.[180] This symptom in the legs and toes is probably due to irritation of the sensory nerve endings. The predilection of the typhoid poison for certain nerves is one of its marked features. In this respect it resembles lead and the diphtheritic poison. The nerves most frequently attacked by it are the peroneal and the ulnar. This paralysis, unlike that of diphtheria, is not likely to follow mild cases of typhoid fever. All instances noted by me have been in severe and protracted attacks of the primary disease. Thus in one instance the patient had been ill for sixteen weeks, having suffered two relapses and several grave complications, including an exhaustive hemorrhage from the bowels. In his case the treatment had been by cold baths for hyperpyrexia, and the suspicion was aroused that this treatment may have been partly instrumental in causing the peripheral neuritis. This possibility, I am convinced, is one of considerable importance, now that the excessive use of the bath is becoming popular. Exposure to cold and wet, we know, is an important contributing cause of multiple neuritis in all cases in which the vitality and nutrition of the nerves are diminished, as, for instance, in chronic alcoholism. Hence there appears to be no good reason why such exposure in the cold bath should not also act in cases in which the nutrition of the peripheral nerve endings is disturbed by the action of the typhoid poison.

Smallpox, as is well known, sometimes causes multiple neuritis. Westphal found disseminated centres of inflammation in the spinal cord in cases of smallpox, and a few observers have noted changes in the nerves. The forms of neuritis seen after smallpox may be local or multiple. Thus the pharynx may be involved, or in some cases a pseudotabes, no doubt due to the involvement of the nerve endings, may occur.

Measles has occasionally been noted as a cause of paralysis, and this paralysis is probably due in some instances to inflammation of the nerves. Ormerod [181] observed three cases of muscular atrophy in one family following measles. The affection invariably began in the legs below the knees. After some months, or even years, it invaded the hand and forearm, and always affected the distal more than the proximal segments of the limbs. He observed trophic lesions resembling chilblains in these cases. The electric excitability of the affected muscles below the knees was abolished and the partial reactions of degeneration were seen elsewhere. From this description it

typhoid type of fever, with sweating and emaciation, and in some instances with a purpuric eruption of the skin. These symptoms were present in the case referred to. While multiple neuritis of wide distribution has not been commonly observed in such cases, there is no *a priori* reason why such an infection of the peripheral nerve endings should not occur. In our patient there was, however, a distinct history of alcoholism, and this factor was probably active, in association with the septic infection, in causing the neuritis. In such a case the general poisoning of the blood by the septic infection of the endocardium may prepare the soil, as it were, for the development of an alcoholic neuritis.

The influence of *malaria* in causing multiple neuritis is a subject about which not much has been written. In the higher latitudes of the United States, where malignant types of malaria are practically unknown, it is not probable that such a complication occurs. Some observers in the tropics, however, have asserted that malarial poisoning causes diffused inflammation of the nerves. Buzzard refers to several cases of peripheral neuritis which occurred in persons who had been exposed to pernicious malarial fever. There was no doubt of the accuracy of the diagnosis. The symptoms, however, were so characteristic of the type of polyneuritis caused by alcohol that Buzzard was inclined to suspect that this was the true cause of the disorder. In one case, in fact, the history of alcoholism was very distinct. If a patient with multiple neuritis comes from a locality in which malaria is rife, he or she will be likely to attribute the symptoms to this poison. Care should always be taken, however, to inquire into the possible history of exposure to alcohol. The characteristic type of polyneuritis, with mental enfeeblement, caused by alcohol should arouse the suspicion of the observer that there was more than malaria acting as a cause. Forchheimer[305] states epigrammatically that there is not a nerve in the body that seems exempt from malaria. He instances the well-known neuralgia of the fifth nerve, and says that the sciatic nerve, the intercostal nerves, and the nerves of the stomach are not uncommonly affected. A rare form of intermittent torticollis due to malaria is sometimes seen, according to this observer, but it is caused probably by irritation of the meninges rather than of the nerves proper. Forchheimer instances no form of multiple neuritis as being commonly seen in malaria.

Among other instances of infectious diseases acting as a cause of multiple neuritis may be mentioned *dengue*. Buzzard refers to such an instance in which there was atrophy and loss of electrical excitability in the tibialis anticus and extensor longus digitorum muscles,

with anæsthesia of the corresponding surface; but as this condition did not occur until more than two years after the febrile attack, it cannot with justice be ascribed to the latter.

Relapsing fever has been observed to be followed by multiple neuritis. Ross (quoted by Buzzard) has published accounts of some of these cases.

One of the most conspicuous examples of the infectious origin of multiple neuritis is furnished by the disease known as *beriberi*. This disease is apparently of infectious origin, that is, it is probably due to some microorganism. It is endemic and contagious. It occurs principally in Asiatic countries, especially China, Japan, and India, but has also been observed elsewhere, as in Brazil. Some authors claim that beriberi is not due to a microorganism, but to insufficient and improper food. Some of the circumstances under which it arises suggest the possibility of a scorbutic taint, but this element is denied by most observers. According to Walker,[""] intestinal parasites are very common in patients suffering with this disease. Thus of 887 patients, 85.5 per cent. had the ankylostomum duodenale. Walker, while he hesitates to admit that this parasite is the real cause of beriberi, thinks that it may be a predisposing factor. It is possible, however, as he suggests, that the presence of the parasite is merely a coincidence, its germs being taken in stagnant water along with the specific germs of the disease. Putnam some years ago reported a series of cases of multiple neuritis in fishermen on the banks of Newfoundland. In these cases the disease seemed to have no cause other than insufficient food and exposure to cold and wet. I cannot but think from some of the accounts of epidemics of so-called beriberi that improper and insufficient food is an active or at least a predisposing factor. A rice diet has been accused of causing the disease, but, considering the vast populations that live on this staple in Eastern countries, some other contributory cause is probably active. Takaki, a Japanese physician quoted by Sinkler,[""] controlled the occurrence of beribier in the Japanese navy by supplying a diet of fresh meat and eggs in addition to rice. Epidemics of multiple neuritis resembling beriberi occur occasionally among sailors during long voyages. In such cases a scorbutic element may be suspected. Instances have occurred in ships carrying cargoes of sugar, and have been ascribed to the carbonic acid gas arising as a product of fermentation. Several patients with symptoms suggesting beriberi were recently seen by me in the Philadelphia Hospital. These men had just arrived from a long voyage in which they had been exposed to privation and hard labor.

Cholera, according to Duplaix, is a cause sometimes of limited

paralyses, which are accompanied ordinarily with disorders of sensation. The clinical aspect of these paralyses is significant, as Landouzy has observed, rather of peripheral than of central lesions. Both the motor and the sensory fibres are involved. From analogy rather than from exact observation, we are led to suppose in these cases the development of a multiple peripheral neuritis.

Among other infectious diseases, *dysentery*, *erysipelas*, and *influenza* do not appear to give origin to peripheral neuritis, or at least if they do so the observations thus far are extremely rare. I have not seen or heard of such instances. It is true that influenza, which has been so extremely rife all over the civilized world in recent years, has been accused occasionally of causing grave nervous complications, among which may possibly be multiple neuritis. When we consider, however, that the tendency has been to ascribe too much to the grippe, we must hesitate before accepting the view that multiple neuritis is one of its sequelæ. Certainly a more exact criticism is required to determine this question. Paraplegia has been ascribed by Landouzy to dysentery. Various forms of paralysis, in fact, have been observed since the time of Galen to follow dysentery (Whitaker). Remak has suggested the idea of an ascending neuritis for this sequela.

Rheumatism has been considered by most writers to be an occasional cause of peripheral neuritis. Gowers and some other writers seem to attribute the neuritis that follows the exposure to cold to a rheumatic humor in the blood. This, however, is entirely hypothetical, and there is no scientific warrant for invoking rheumatism as the cause under these circumstances. It is indubitable, however, that in cases of acute and inflammatory rheumatism involving the joints peripheral neuritis occasionally occurs. John Hunter was probably the first to note the fact that an inflammation of a joint frequently causes rapid wasting of the muscles that move that joint, and that this wasting is not the result of inaction, but is an active process, and is frequently accompanied with paralysis. Such muscular atrophy may even accompany non-traumatic synovitis, as was observed by Duchenne. Vulpian, as most pathologists of his time, attributed to these paralyses a reflex origin. Valtat in his thesis reviewed this whole subject, and called attention to the fact that the paralysis may precede the atrophy in the affected muscles. He found that the extensor muscles were the most profoundly affected. In experiments on dogs he injected irritating substances into the joints, and thus caused intense synovitis, which was followed by rapid and extensive atrophy, which might even involve the whole limb, although the extensor muscles were most involved. Valtat, however, over-

looked the true nature of the lesion; as he found no signs of inflammation in the affected muscles, he adopted Vulpian's theory of reflex origin. This whole subject has been reviewed by Bury,[907] who concludes that in articular rheumatism we meet with muscular atrophy and paralysis, which he is inclined to ascribe to organic changes either in the central or peripheral nervous system. He also calls attention to the fact that peripheral neuritis may occur during convalescence from rheumatic fever, in a limb in which there is no involvement of the joint. This latter observation is of the utmost importance, as it shows conclusively that the rheumatic poison circulating in the blood may be the direct cause of inflammation of the peripheral portions of the nerves. From accumulated evidence it seems possible that the wasting and paralysis in direct association with inflamed joints are due to the action of some septic substance thrown upon the nerves in the neighborhood of the joints. This would explain the fact that not only in rheumatism but also in traumatic and septic forms of synovitis, this wasting and atrophy of muscles occurs. While as a rule the muscles in connection with the joints are the first and the most involved, yet in many instances muscles remote from the joints may suffer; this, of course, is still further in accord with the view that the active agent is a poison circulating in the blood. With reference to the presence of a rheumatoid poison in the blood in cases in which cold alone has acted as a factor, and in which the symptoms of acute rheumatism are lacking, I can only say that such cases are entirely distinct from those here under consideration.

Arsenic, as is well known, may cause peripheral neuritis. Symptoms of paralysis in arsenical poisoning have been noted for many years. Seguin[908] gives references to this subject dating back as far as the thirteenth century. Beck, in 1838, noted a variety of arsenical poisoning, in which there was first an inflammatory action followed by a second stage, that of nervous involvement. The nervous symptoms varied "from coma to a perfect palsy of the arms and legs." Gibb [909] records the case of a lady who had taken arsenic, mostly Fowler's solution, for many years for a skin affection. She suffered with neuralgic pains in the groin, shoulders, and sides, followed by loss of power in her lower limbs. McCready[910] relates a case in which a woman rubbed white arsenic mixed with gin on the head of her child, suffering with favus; the child died in less than two days, with its legs completely paralyzed. Popoff, of St. Petersburg, undertook an experimental study of arsenical paralysis; the lesions were produced by administering the poison to animals. He concluded that arsenic causes acute central myelitis, and the peripheral nerves remain normal even three months after the ingestion of the

drug. These conclusions, however, are not in accord with those of other observers. Mills[111] has reported a series of cases of arsenical poisoning caused by eating pumpkin pie, in which a large amount of arsenious acid had been placed. In these cases the symptoms of peripheral neuritis were well marked. These symptoms were pain in the legs below the knees, paræsthesia, and loss of power. There were also atrophy and the reactions of degeneration. In recent years cases of arsenical paralysis have been noted as a result of the administration of this drug in large doses for chorea. Cases of this kind have been reported by Potts, from the Hospital of the University of Pennsylvania. This constitutes, in fact, a well-defined risk in the administration of large doses of arsenic for this disease. Workmen in arsenic are, of course, exposed more or less to the effects of the poison. Among these workmen miners of tin, copper, and nickel ores, and artisans who work with Scheele's green and Vienna green, may be mentioned; also paper-hangers and workmen employed in the manufacture of artificial flowers. Arsenic occurs in some of the aniline dyes. Putnam reported an epidemic of arsenical poisoning among the children in an infant asylum, which was caused by arsenical dyes in some dress-goods.

Mercury occasionally causes a peripheral neuritis; this may be either local or diffused. Intense pain is a not uncommon symptom of chronic hydrargyrism as observed among the workmen in the Spanish mines at Almaden. This begins in the extremities, usually in the great toe or thumb, and spreads upwards from these points. The pain is intense and lancinating, and is followed by loss of power. Among local palsies are those of the extensors of the wrist and of the muscles of the larynx. These were noted long ago by Vicente de Arevaca.

Pneumonia, especially in children, as is well known, may present grave complications in the nervous system. Among the most common of these is hemiplegia. Duplaix states that there is in pneumonia a form of paralysis the evolution of which recalls sometimes diphtheritic paralysis, sometimes Landry's disease. He suggests the hypothesis of a peripheral neuritis for these cases.

Diabetes, as is well known, is complicated with a number of accidents in the nervous system. The most conspicuous of these, of course, is diabetic coma. A not uncommon disorder, however, in diabetics is a severe type of neuralgia. This is sometimes associated, especially in the legs, with cramps. This form of diabetic neuralgia has been recognized for many years. Its favorite seats are in the sciatic, peroneal, and inferior dental nerves. Worms studied these neuralgias in 1881 and did much to secure their clinical recog-

nition. Cornillon in a study of twenty-two cases tried to establish the theory that the neuralgia in diabetic patients is as much due to uricæmia or a gouty diathesis as to hyperglycæmia, and that the condition is not due to neuritis but to transitory changes in the nerve centres (Tyson). But this theory is excluded by the facts, now well established, that these neuralgic pains in diabetes are frequently the forerunners of well-marked motor paralysis, and that many of these cases present unmistakably the features of peripheral neuritis. In a case recently under my care in the Methodist Hospital, Philadelphia, the patient, a woman aged sixty-one years, had marked symptoms of peripheral neuritis, coming on in a few weeks after the onset of glycosuria. The type of paralysis in this case was pseudotabes, which will be described later. The various disorders in the nervous system, caused by diabetes, have been ascribed by some to the presence in the blood of acetone, which is a product of the decomposition of sugar. More recently, however, the poison has been thought by others to be an intermediate product between sugar and acetone—in other words, an acetone-producing substance, as acetoacetic acid.

The influence of the *puerperium* in causing neuritis has already been referred to in the section on diseases of the sacral plexus. This influence is no doubt exercised by means of sepsis; in other words, there is introduced into the blood from a septic metritis or pelvic cellulitis a poison which acts directly upon the nerve trunks or their terminal branches. In the case of the nerves within the pelvis, however, this inflammation extends by direct contiguity. Whether or not cases may arise of a more widespread multiple neuritis due to sepsis following childbirth, is perhaps an open question. Some observers have put on record cases of alcoholic multiple neuritis occurring in childbed. It would seem almost as though women who had reduced themselves by excessive indulgence in alcohol were rendered more liable to the outbreak of neuritis by the exhaustion from hemorrhage, etc., in childbearing. The explanation for this probably is that the nutrition of these patients is already depraved by the excessive use of alcohol, and that the prostration of strength due to the pain and hemorrhage caused by their labors prepares the system for a manifestation of the activity of the poison. Hence care should be taken in all these cases to determine whether or not there has been exposure to alcohol. The occurrence of delirium tremens, as I have pointed out elsewhere,[111] as an accompaniment of the puerperium presents a subject of some interest and importance in the diagnosis, and the same may be said of the occurrence of peripheral neuritis in childbed. In women addicted to drink, pregnancy may be an additional incentive to it, and the indulgence will usually, of

course, be secret. The puerperium, with its depression and exhaustion, may then precipitate quite unexpectedly an attack of neuritis. Copland[1,2a] was one of the first to call attention to the explosion, as it were, of alcoholic complications in the puerperium. He had reference, however, more particularly to the outbreak of mental symptoms. But from my own observations I am convinced that symptoms of peripheral disease may also occur suddenly under these circumstances. I have under my care now in the Philadelphia Hospital a young married woman who recently miscarried in the third month of her pregnancy. Within two weeks well-marked symptoms of peripheral neuritis, of distinctly alcoholic type, set in. The patient confessed to a daily indulgence in beer and whiskey. She is a thin, anæmic, underfed woman, who has been much exposed to cold and wet, and has done much hard work. In her miscarriage she lost enough blood to prostrate her still further, and this apparently precipitated an attack of peripheral neuritis. She has no signs of sepsis.

The influence of *cold* and *dampness* in the causation of peripheral neuritis is an important one. It is not an easy one to explain according to our present ideas of a humoral pathology. Gowers, as already said, has suggested that under the influence of cold a rheumatoid poison is generated, and that this causes an inflammation of the nerve trunks or their terminal branches. But I can see no warrant for adopting such an hypothesis. In these cases cold and wet seem to me to act largely as contributory causes. When the system, for instance, is surcharged with alcohol or some other poison in the blood, such exposure, by diminishing in some way the resisting power, may be the immediate cause in precipitating an attack of neuritis. It does this probably by diminishing or altering in some way the blood supply to the peripheral nervous system, the vitality of which being thus lowered, these delicate nerves feel the more easily the poisonous effects of the alcohol. I have seen many such instances. In fact, it is not uncommon in my observations at the Philadelphia Hospital to see patients whose attacks of peripheral neuritis were immediately caused by hard work and by exposure in the midst of cold and dampness. Whatever the theoretical explanation may be the clinical fact is indubitable.

The influence of *sex* in the causation of multiple neuritis is only secondary and contributory. Women undoubtedly suffer rather more frequently from alcoholic multiple neuritis than do men. This is probably due to several causes. Thus women have probably less resisting power, owing to a more delicate structure of the nervous system, than have men. Moreover, in the class of women in which

the disease is most common, the patients are not infrequently reduced by household drudgery, poor food, and over-childbearing. Under these circumstances some women form a bad habit of constant tippling, taking small quantities of whiskey or beer at short intervals during the day, and as a consequence not eating a sufficient quantity of food. They thus lay the foundation for the disease in its most aggravated type.

The influence of *race* in determining multiple neuritis is not marked.

Among rarer causes of multiple neuritis may be mentioned *oxide of carbon* and *sulphide of carbon*. Leudet has described a neuritis due to carbonic oxide. The symptoms may appear immediately after the exposure or may not develop for some days. The paralysis may be limited to one limb but usually extends to others. Sapillier has demonstrated that poisoning by sulphide of carbon may cause paralyses which attack by preference the flexors of both the upper and lower limb. *Phosphorus* also has been observed to cause neuritis. In *ergotism* there is sometimes an involvement of the sensory and motor nerves.

Symptoms.

The symptoms of multiple neuritis are motor, sensory, trophic, visceral, psychic, and those that are manifested in the special senses.

The Motor Symptoms.—The motor symptoms may be subdivided into five classes, viz., paralysis, cramps, tremor, ataxia, and contracture. I shall describe these in the order given.

Paralysis is, of course, one of the most common and striking characteristics of peripheral multiple neuritis. It may be said, indeed, to be almost a constant symptom, since there are few cases in which it is not seen; but it must be remembered that it is not absolutely constant, since in one class or type of cases—*i.e.*, the ataxic or pseudotabetic variety—it is not necessarily seen.

In order to understand the type of the paralysis present in multiple neuritis it is desirable to recall its exact mechanism. This paralysis is caused by an interruption or destruction of the axis cylinder of the peripheral motor neuron, which, as already explained, has its cell body or trophic centre in the anterior cornu of the gray matter of the spinal cord. Not only the power to move the muscle but also the tone, the electrical reactions, the reflexes, and the nutrition of the muscular mass depend upon the integrity of this peripheral motor neuron. This multiplicity of physiological functions in the muscle dependent upon the neuron must be clearly understood by the student of nervous diseases in order that he may

obtain an adequate and systematized idea of the kind of palsy seen in multiple neuritis. In other words, he must realize that the paralysis is only one of several associated symptoms, and that this characteristic association of symptoms is seen under no other circumstances of disease in the body. As a consequence of this association the paralysis of multiple neuritis is of the flaccid and atrophic variety. The muscular tone is markedly lowered and the nutrition of the muscles suffers promptly. This results in the muscle becoming flaccid and soft, and losing weight and bulk exactly proportionate to the number of its neurons involved and the completeness of their impairment. With this wasting and lowering of the muscle tone there occur promptly alterations in the response of the muscle to electricity and an abolition of its tendon reflex. Thus the alterations in the muscle in peripheral multiple neuritis are paralysis, flaccidity, atrophy, loss or alteration of its electrical reactions, and loss of its proper tendon reflexes. While these are the five cardinal points in the muscular phenomena of multiple neuritis, it must not be supposed that they are all equally distinct in every given case. Their association varies somewhat according to circumstances, *i.e.*, according both to the extent of the inflammatory process in the neuron and also to the individual poison acting to promote this inflammation. In minor degrees of inflammation, for instance, the motor paralysis may not be complete, and yet in these cases, after a few days, the response of the muscle to faradism may be abolished. Again, in some cases, although the paralysis may be promptly established, the atrophy of the muscle does not advance to a marked stage before a tendency to recovery stays its progress. In such cases the muscular tonus may not be absolutely impaired. As a rule, however, it may be said that the order of these phenomena is as follows: The first symptom manifested is paralysis. Next is diminution or abolition of the faradic contractility, to be followed later by flaccidity sufficiently advanced to be recognizable; and then, later still, by perceptible atrophy. The order of occurrence of impaired reflexes varies somewhat. According to the observations of some writers, the reflexes at first may be even slightly exaggerated, to be followed by gradual diminution and ultimate abolition. In my observation, however, exaggerated reflexes, even in the earliest stages, are practically unknown. I cannot recall an instance.

The individual poison acting to cause multiple neuritis may present some characteristics that distinguish it from other agents. This, however, is with reference not so much to the degree as to the distribution of the multiple neuritis. As a rule, however, these toxic agents manifest some special characteristics, even in their mode of

attack. Thus alcohol often acts with extreme malignancy. Before its onslaughts the neuron and the muscular tissue sink rapidly and all the motor features of peripheral neuritis, as just enumerated, show themselves with extreme rapidity. This is all the more noteworthy because in these cases the alcohol has been taken, as a rule, during long periods of time before it suddenly breaks loose, as it were, upon the peripheral nervous system. Lead, on the other hand, is usually rather more gradual in its attack. The atrophy is especially slow, as a rule, in appearing and the reactions of degeneration may not be complete until the paralysis or paresis has lasted for a comparatively long time. These cases vary, however, and some are more explosive than others. In cases of diphtheritic paralysis the onset is often quite unexpected and after convalescence has been apparently satisfactorily begun. In these cases the motor weakness is very marked from the beginning, but as many of them tend to recover promptly with proper care and treatment, they do not advance, as a rule, to marked wasting and degeneration of the muscles. Hence in mild cases with considerable paralysis there may be only a partial reaction of degeneration.

The distribution of the paralysis in peripheral neuritis varies exceedingly. As a rule, however, the paralysis is usually symmetrical, *i.e.*, appears in corresponding groups of muscles on both sides. This is one of its most marked characteristics, and is due, of course, to the fact that the poison circulating in the blood is carried equally to the nerve trunks and their terminal branches on both sides. This serves to distinguish multiple neuritis from many forms of paralysis due to central disease, especially diseases of the brain.

Another characteristic is the tendency of the extensor muscles to suffer more than the flexors. This gives rise to some of the most noted features of multiple neuritis, such as the wrist drop and foot drop, that are so commonly seen. These symptoms are caused, of course, in the case of the hand and forearm by paralysis of the extensor of the wrists and fingers, and in the case of the feet by paralysis of the peroneal group, the tibialis anticus, and the extensors of the toes. In estimating this paralysis of the extensors, an important physiological law must not be overlooked, namely, that the contraction of any muscular group that assumes, in any coördinate movement, the principal rôle is dependent upon the integrity of other and often antagonistic groups. It was demonstrated, especially by Duchenne and Winslow, and has been confirmed universally by clinicians, that in every volitional movement antagonistic muscles contract simultaneously. It thus happens, for instance, that the movement of flexing of the wrist and fingers demands, in order to be executed normally, a

simultaneous contraction, more or less strong, of the extensor muscles (Babinski). Therefore in paralysis of the extensor muscles of the hands and feet, so marked in multiple neuritis, there often appears to be more or less impairment of power in the flexor muscles of these parts. In lead palsy, for instance, in which wrist drop is often absolute, the patient usually has a much diminished power or grip in the hands. This movement is not only impaired in power, but it is also performed in an awkward or incoördinate manner; but this, for the reason already explained, is not an evidence of true paralysis in the flexor group of muscles. In very advanced cases, however, even the flexor muscles may be involved, as is shown by their flaccidity and the reactions of degeneration.

Another characteristic of multiple neuritis is the tendency of the distal muscles of a limb to be paralyzed to a greater degree than the muscles whose points of origin are in or near the trunk. Hence, in the arm, the muscles of the hand and forearm are more likely to be completely paralyzed than the muscles of the upper arm and shoulder, and in the lower limb, the muscles of the leg and feet are more paralyzed than those of the thigh and hip. This is in accord with the fact that the poison causing multiple neuritis expends its energy not only in the peripheral distribution of the nervous system, but also in the peripheral distribution of the blood-vessels. This law of distribution seems to indicate that the axis cylinders of the peripheral motor neurons are the more likely to suffer from the effects of a poison in the blood the farther they extend from their trophic centres—the cell bodies of the neurons. This clinical fact does not support the view of Marie and others, that the axis cylinder of a neuron draws all its nutritive supply from its cell body; but it indicates that the axis cylinder is nourished in its course possibly at the points of constriction between the nodes of Ranvier. If this latter view is correct, it would follow that the axis cylinder at its periphery may for some reason feel the direct devastating effects of a poison in the blood, while its more central portion and its cell body itself entirely escape. The clinical fact, whatever may be its explanation, is as here stated. In these cases, consequently, the patient presents the appearance of distinct peripheral palsy, marked not only in the extremities but in the distal segments of the extremities. There may, it is true, be cases of multiple neuritis of irregular distribution in which this appearance is not well marked, but in the majority of cases the rule holds good. Thus these cases present a characteristic clinical appearance which serves to distinguish them from cases of paralysis due to other lesions.

In the upper limb the paralysis, according to the rule laid down,

affects the muscles of the hand and forearm more than those of the upper arm and shoulder, and the extensor muscles more than the flexor. In the ordinary type of case the muscles most involved are the extensor carpi radialis, extensor carpi ulnaris, extensor communis digitorum, and the extensors of the index and little fingers. The extensor proprius pollicis is not always so markedly involved. As a consequence of this paralysis the attitude of the arm and hand is as follows: There is the characteristic wrist drop, the hand being held in the attitude of pronation or semipronation. If the hand and arm are placed upon a horizontal plane, the former is seen to be slightly adducted. As a rule, all the phalanges are not equally paralyzed; the second and third may be still slightly extended. Usually the phalanges of the thumb cannot be extended, although this rule is not absolute. Abduction and adduction of the hand are weakened or abolished. As already explained, the flexor muscles, even though not paralyzed, cannot contract and perform coördinate movements in a normal way. This is due to the fact that normally coördinated movements require the activity of antagonistic groups of muscles. It is a mistake, however, to suppose that the flexor muscles are always absolutely exempt in multiple neuritis. In aggravated cases they too are involved, and altered electrical reactions prove this. They are never, however, involved so early or so markedly as the extensors; and consequently, when contractures take place later in the case the limb is always held in a position of partial flexion. Involvement of the muscles of the upper arm, while not as a rule so marked as that of the muscles of the forearm and hand, is yet nearly always seen to some extent in multiple neuritis. The location and degree of the paralysis vary, of course, according to the nature of the case. Not uncommonly the paralysis presents the so-called brachial type in which the group of muscles originally pointed out by Duchenne is involved. These muscles are the deltoid, biceps, brachialis anticus, supinator longus, and occasionally the infra- and supraspinati and the pectoralis major. These muscles seem to constitute a physiological group, which, as is well known, can be thrown into conjoint activity by faradizing the brachial plexus at a point just above the clavicle. In multiple neuritis they are usually associated in the paralysis. As a consequence the attitude of the arm is quite characteristic. The arm lies against the trunk, rotated inwards, with the forearm in a position of semipronation (Fig. 41). The patient is unable to abduct the arm from the trunk or to rotate it outwards. Moreover, the forearm cannot be flexed at the elbow, but if passively flexed it can be extended by the patient. Occasionally this type of paralysis is not absolute. One or other of these muscles may

be paralyzed more than the other members of the group. The deltoid is perhaps especially apt to suffer. In advanced cases or cases of an aggravated type, the extensor muscles on the back of the arm may also be involved.

A special type of paralysis of the arm and hand, called the "Aran-Duchenne type," has been described. In this type the paralysis and atrophy are usually marked in the hand, especially in the thenar and hypothenar eminences, the interossei muscles, in particular the abductor indicis. This type usually begins in these small muscles of the hand, and is slowly progressive. Later it involves the deltoid, the extensors and flexors of the wrist, the supinators, the biceps, and the triceps. In this type the paralysis usually begins on one side and

FIG. 41.—Alcoholic Multiple Neuritis. (Philadelphia Hospital).

passes eventually to the other. This type of paralysis is not common in multiple neuritis. While it is probably due to some form of degeneration or inflammation of the motor neurons, it cannot properly be classed with the ordinary cases of the disease here under consideration.

In the lower limb, as in the upper, the paralysis is most marked in the muscles the most remote from the trunk. Thus the muscles below the knee are the earliest to suffer, and in most cases continue all through the case to be the most involved. The muscles especially affected are the peroneal group, the extensor longus digitorum, the extensor proprius pollicis, and the tibialis anticus. The paralysis of these muscles permits the foot to assume the characteristic posi-

tion known as foot-drop. This is strictly analogous to the hand-drop already described. In this attitude the foot drops until it is almost or quite in a straight line with the leg. The power of extension is usually absolutely abolished. The phalanges of the toes at the same time are flexed, and the patient usually has no power whatever to move the toes. This loss of power in the extensor muscles causes a characteristic gait, when the patient is still able to walk. Thus the patient, because of his inability to extend the foot, is obliged to lift it very high in order that the toes may clear the ground. As he does this, the foot hangs apparently as an inert mass and when it comes down strikes the ground first with the toes, in marked contrast with the gait of tabes, in which the heel first strikes the ground. This gait is known by the French as the "gait de steppage." It has been graphically called "the turkey gobbler walk." Moreover, the outer border of the foot drops, so that when the patient puts his foot upon the ground he strikes with the external border first. This is due to paralysis of the peroneal muscles. As in the case of the arm, however, the extensor muscles are not the only ones paralyzed in aggravated types of multiple neuritis. The sural or calf muscles may also be involved. They become flaccid and atrophied, and present the reactions of degeneration. As in the arm, however, they are not so early or so completely paralyzed, as a rule, as the extensor muscles. Babinski calls attention to the fact that in rare cases paralysis of the triceps muscles may be most marked. The position of the foot is then different from that already described. In place of falling, it is raised at a right angle to the leg, and the movement of extension (or dorsiflexion) is preserved. In walking the patient then, at the moment of placing his foot upon the ground, strikes first with the heel, resembling in this respect a patient with locomotor ataxia. Babinski states that he has seen an example of this gait in a patient suffering with peripheral neuritis. In this case, however, there was an associated paralysis of the peroneal muscles, permitting the external border of the foot to fall. The thigh muscles are usually not exempt in peripheral neuritis, although, as a rule, they are not involved so early and so completely as the muscles of the leg. The quadriceps extensor is often paralyzed and atrophied, and may present the reactions of degeneration. The flexor muscles on the back of the thigh are usually not so much impaired and in course of time they become contractured and hold the leg partly flexed at the knee. This contracture of the hamstring muscles constitutes one of the embarrassing features of aggravated cases of multiple neuritis.

The muscles of the trunk are not exempt in multiple neuritis.

One of the most serious complications of the disease is paralysis of the muscles of respiration. In some cases this is so marked that the expansion of the chest is seriously impaired, or even, as I have seen it in the upper part of the chest, entirely abolished. This is probably due in part to involvement of the long thoracic nerve, which supplies the serratus magnus muscle. The phrenic nerve also may be paralyzed in some forms of multiple neuritis. This involvement of the respiratory muscles constitutes a grave risk to these patients. The interference with the proper expansion of the lungs leads to a catarrhal state of the bronchial mucous membrane, and may even induce pneumonia. In these cases this complication is almost necessarily fatal. This is so especially when the heart muscle is involved, as sometimes happens; this will be described later. The paralysis of the muscles of respiration may be determined by placing a tape measure around the chest and bidding the patient take a long breath. In grave cases it will thus be found that the expansion of the chest, which is normally to the extent of from two and a half to three and a half inches, is reduced to probably one inch or may even be abolished entirely. This examination should never be neglected in the study of these cases.

The muscles of the head, face, and neck are not involved, as a rule, in peripheral neuritis. The facial muscles have been seen to be paralyzed in rare cases. The muscles of deglutition, however, are impaired notably in the type of peripheral neuritis caused by the diphtheritic poison. This is never so, however, in cases due to alcohol. The trapezius and sternocleidomastoid muscles also are not involved in the ordinary type of multiple neuritis. In diphtheritic paralysis, however, the head may drop forward because of paralysis of the trapezius muscle. The muscles presiding over the functions of the bladder and rectum also escape in this disease. In rare cases the vocal cords are paralyzed. This is probably due to involvement of the pneumogastric nerve, which also causes the tachycardia not infrequently seen in these cases.

The occurrence of *cramps* in multiple neuritis is probably not so rare as is indicated by the fact that few if any writers refer to the subject. In my own observations I have attempted, sometimes in vain, to elicit a history of cramps from patients or their friends, but I am convinced that this symptom is not uncommon. My attention has often been called to the subject by a statement of a patient, particularly in cases of alcoholic multiple neuritis, that he or she had suffered with cramps in the legs in the early stages of the disease. I have myself seen some marked manifestations of this symptom, but have had to rely in many cases upon the statements of the patients

themselves. In such cases, of course, the severe pains in the legs might possibly be erroneously called cramps by a patient whose mental faculties were blunted by chronic alcoholism. There seems to be no *a priori* reason, however, why cramps should not occur in the very early stages of peripheral neuritis. The poison at this stage might act first as an irritant, sufficiently powerful to excite cramps or painful contractions in the muscles. Vulpian and others demonstrated that pressure on a nerve trunk induces cramps in the muscles supplied by that particular nerve. This is an early symptom, and seems to indicate a reaction of the neuron to an irritant lesion. Later, of course, after the vitality of the neuron is lowered and its function is impaired or suspended, cramps are not possible. This manifestation of the hyperexcitability of the neuron and its muscle in an early or irritant stage of neuritis might be looked upon as analogous to the hyperexcitability of the muscle to electricity, which we know is seen as an early phenomenon of the reaction of degeneration.

As will be seen later, in the section on contracture, Babinski claims that cramps are not uncommon in multiple neuritis, but he applies this term evidently to the more or less prolonged and sustained contractures which usually occur in the early stages of the disease. These early painful contractures or cramps are rather more persistent and rather less painful than that state of a muscle which is usually denominated "cramp." They may, however, be overcome by passive extension, and, as they cease to be painful as soon as the muscle is thoroughly extended or relaxed, they bear undoubtedly a close analogy to a true cramp. Moreover, as they occur before the muscle has degenerated, they are presumably due to a contracture of true muscular tissue and not to a mere retraction of the fibrous tissue of a degenerated muscle.

Tremor has been noted by many writers in cases of multiple neuritis. It is not uncommonly seen in alcoholic subjects. This tremor is usually of the vibratory type (*i.e.*, of rapid oscillations), and is much increased by voluntary movement. It is a symptom seen also in poisoning by lead and by mercury. It is not always easy, however, to eliminate from these cases the etiological factor of hystesia. The toxic hysterias, as, for instance, the hysteria due to lead or to alcohol, present some difficult problems. It is common to attribute all the patient's nerve symptoms to the poison from which he suffers; whereas there may be a grave psychical element, which acts in its turn to produce symptoms simulating organic disease. Few clinicians can have failed to observe hysterical symptoms in chronic alcoholics; and this same class of symp-

toms may be seen occasionally in cases of poisoning by lead and mercury. Pitres[113] reports a case of hysterical tremor following delirium tremens. The patient had other hysterical symptoms, and his tremor was cured with a magnet. Letulle[114] has described the hysterical symptoms supervening in the course of chronic mercurial poisoning. Among these were hemitremor, sometimes transferable from one side to the other. The tremor of mercury has been stopped by faradism, or by causing the patient to squat and walk in this position, although when erect he could not walk for the tremor. The fact seems to be, as I have said elsewhere, that the metallic poisons, as well as alcohol, can excite hysterical symptoms in predisposed persons; and among these symptoms one of the commonest is tremor. The fact of alcohol being a well-known hysterogenous poison is a presumptive proof of the same power in lead and mercury, and especially with reference to tremor. But while the various poisons that cause multiple neuritis can also excite an hysterical tremor, they do also indubitably in many cases excite a tremor which is organic. The necessity for distinguishing these two types of tremor is very great, and can best be done by noting the influence of suggestion and the presence or absence of other and confirmatory hysterical stigmata. It is also a question in these cases whether a tremor, even of organic origin, can properly be looked upon as due to a neuritis, even though it coexists with the other symptoms that are caused by neuritis. Tremor, in fact, is probably due to impairment of the nerve centres rather than of the nerves themselves, and if it exists along with the symptoms of neuritis, is rather a coincidence than otherwise. In fact, as the symptoms of neuritis become more and more pronounced, the tremor, in my observation, is likely to disappear. It seems then to be gradually supplanted, as it were, by the symptoms that indicate an impairment of the transmitting power of the axis cylinders. This is what we should expect if tremor is due to an incoördinate action of the nerve centres. This could only show itself so long as the axis cylinder was able to transmit impulses, even though these impulses arose from incoördinating centres. As soon as this transmitting power became impaired by inflammation and degeneration of the peripheral portions of the axis cylinders, no impulse could pass; and consequently the tremor would be supplanted by a flaccid paralysis. It thus seems that, strictly speaking, tremor cannot be regarded as a symptom of peripheral neuritis. It is rather a symptom, independent and preëxistent, of the poisonous action of the substance which has also caused the neuritis.

Ataxia, or incoördination, is a not uncommon symptom of pe-

ripheral neuritis. It characterizes, indeed, a distinct type of the disease, namely, the sensory type, or the so-called "pseudotabes." I have reported cases of this form of peripheral neuritis in a paper already referred to.[17] In 1884 Déjerine[216] described a disease which he called *nervotabes périphérique*, and which was practically the ataxic type of peripheral neuritis. His paper was founded upon the clinical histories and autopsies of two patients who had been supposed to have locomotor ataxia. The symptoms in these patients, both of whom had used alcohol to excess, were incoördination, anæsthesia, abolition of the knee jerks, atrophy, and slight paresis, without symptoms of involvement of the eyes or bladder. At the autopsies changes were found in the cutaneous nerve endings, and to a less extent in the intramuscular nerves, and no changes whatever in the spinal cord, nerve roots, or ganglia. Déjerine was probably the first to call attention to this form of multiple neuritis, and deserves the credit of demonstrating the striking resemblance of these obscure cases to locomotor ataxia. Dreschfeld [215] about the same time described a type of alcoholic ataxia characterized by incoördination and loss of knee jerks with lancinating pains, but without atrophy or paralysis. Krüche[217] also wrote a paper on "Pseudotabes in Alcoholics." These observers had only noted symptoms which had been seen and described before, notably by Wilkes[196] and Leudet,[218] but which had not been ascribed to their true cause. The latter writer, for instance, had described the same combination of symptoms, anæsthesia, hyperæsthesia, disordered gait, and paresis, and thus came near to describing what we recognize to-day as the ataxic type of polyneuritis. This type was still more clearly differentiated by Leyden, in 1888, under the head of acute ataxia, for which he borrowed from the earlier paper of Déjerine the title of "neurotabes peripherica."

It may be said in general terms that all these cases of pseudotabes or acute ataxia are instances of the sensory type of multiple neuritis as described by Leyden, and that their pathological anatomy is probably that originally described by Déjerine, namely, involvement of the sensory nerve endings with a slighter degree of inflammation in some cases of the intramuscular nerves. Hence the common symptoms are anæsthesia, hyperæsthesia, ataxia, and abolition of the knee jerks. In not a few of these cases there is also some degree of paresis and muscular atrophy, although these often require close observation to determine them. From this pure sensory type, with ataxia, all degrees of severity occur up to well-recognized forms of multiple neuritis with general involvement of the sensory and motor nerves. This type in its purity closely resembles locomotor ataxia, although the eyes and the bladder are not involved. This exemption consti-

tutes an important point for differentiation. Paralysis may be so little marked in these cases that when the patients are lying down they have comparatively good control of the limbs, but on attempting to walk the ataxic gait becomes at once evident. This ataxia is no doubt due to involvement of the peripheral sensory neurons, and hence is strictly analogous to that which occurs in locomotor ataxia. The difference in the pathology of the two affections as regards the sensory neurons is simply in the location of the disease process. In multiple neuritis the inflammation is localized in the terminal branches and arborizations of the sensory neuron in the skin, while in locomotor ataxia it is localized in the ganglia of the posterior root and in the extension upwards of the sensory neuron in the posterior column of the spinal cord. The gait of these patients closely resembles that of locomotor ataxia. There may be the same flapping gait, in which the patient brings his heel first to the ground, as in tabes. If, however, as not unusually happens, there is some paresis of the extensor muscles of the foot and toes, the patient may have some degree of foot-drop, and may then present the high-stepping gait, already described, in which, in order to clear the ground, he is obliged to lift the foot very high, and strikes the ground first with the toe. These patients also sway violently with the eyes shut, and have the same difficulty in walking in the dark that is experienced by a tabetic. They also, as a rule, have a sense of numbness in the feet, which causes them to feel as though they were standing or walking on some soft substance. The true explanation of this ataxia is no doubt to be found in the affection of the sensory neurons, just as in tabes. There is abolition of the muscular sense and of the sense of location and distance necessary for the proper coördinating of the movements of the feet and legs. This ataxic type of multiple neuritis may occur not only as a result of alcohol, but also of lead and diphtheria. In one case, which I have reported, the patient had been exposed for many years as a painter to lead, and he had also indulged freely in alcohol.

Contracture is one of the most important symptoms of multiple neuritis. It is usually, but not necessarily always, a late symptom. It is most marked in the leg, especially in the flexor muscles on the posterior parts of the thigh. Contracture of these hamstring muscles causes a partial flexing of the leg at the knee, so that as the patient lies in bed in the dorsal position the knees are usually slightly elevated and the feet rest upon the heels (see Fig. 41). This is quite a characteristic attitude in peripheral neuritis. These contractures are usually the cause of much pain and distress to the patient. In some cases the slightest movement or attempt at extension, either active or

passive, causes cries of distress or expressions of pain on the face. In the early stages the contracture may usually be overcome by firm persistent efforts at extension by the physician or attendant. The pain caused by this passive extension seems to be relieved as soon as the legs are completely straightened out. When left to themselves, however, the legs quickly assume the position again of contracture. That contractures are not necessarily late phenomena in multiple neuritis, I have had ample opportunity to verify. In fact, I think a tendency to contracture of the hamstring muscles is very early witnessed in most cases of the disease. In the early stages these contractures, as already stated, may be overcome with passive motion; but in later stages they become more set, so that complete extension of the legs may become quite impossible. In a case recently examined by me, in a young woman suffering with an acute form of alcoholic multiple neuritis, the contracture of the flexor muscles of the legs was well marked, although the patient was only in the third week of the disease. In her case they could be overcome by firm and persistent efforts at extension, but these attempts caused her acute pain, of which she complained bitterly. Other muscles or muscle groups besides the flexors of the leg may present contractures. In fact, as these cases advance, most of the flexor muscles, being unopposed by the paralyzed extensors, pass into a state of more or less persistent contracture. The sural group of muscles, for instance, may be in this condition. Thus the foot is not only dropped, but it is held extended by a contracture of the muscles attached to the tendo Achillis. This may cause a rigid extension of the foot, which in advanced cases cannot be overcome even by passive motion. In the arms the muscles most affected by contracture are the flexors of the hand and wrist. There is usually little if any contracture of either the flexor or extensor muscles of the upper arm, but the tendency here also is for contracture of the flexors to preponderate, thus causing slight flexion of the arm at the elbow.

These contractures in multiple neuritis appear, as just described, in the flexor or least paralyzed group of muscles. That they are partly due to the unopposed action of the muscles involved is evident. As these muscles, however, are probably never entirely exempt from the action of the poison, it would probably be erroneous to claim that the contracture was entirely a negative, rather than an active, result of the disease. That they are in some way due to the direct action of the poison upon the neurons or the muscular fibres, or both, seems to be indicated by the acute symptoms which they present in many cases. These symptoms especially are the pain, not only on effort at extension, but also on pressure or even gentle handling, of the

contractured muscles and their tendons. This severe pain on handling would scarcely be witnessed in a normal muscle or tendon. If careful investigation is made in these cases, it will be found usually that the hamstring tendons and muscles are hypersensitive to the slightest touch, especially in the early stages of the disease. Later, after the contractures are well established, this tenderness is not so marked. Moreover, these muscles, while not so severely paralyzed as the extensors, are in no case exempt, since they are distinctly paretic and atrophied, and present reactions of degeneration.

With reference to the exact pathological condition underlying these contractures, some questions may be raised. It depends somewhat, of course, upon the significance attached to the word "contracture," whether or not this condition in multiple neuritis may come properly under this term. The word in its simplest meaning indicates, of course, a more or less permanent or sustained rigidity and shortening of the muscles, due to a physiological contraction of its true muscular fibres. This is what happens probably in the contractures of hemiplegia, paraplegia, and hysteria, in which diseases the muscle is not undergoing a true degeneration. But the conditions are not the same in peripheral multiple neuritis. In this latter disease the muscle is degenerating, and its true muscular fibre not only soon loses its power of contraction to the electrical currents, but even loses its structural identity. Consequently it cannot be affirmed with reason that the contractures, especially the late contractures, of multiple neuritis can be due to a physiological contraction or over-action of the muscular fibres, which no longer exist. Babinski[1] criticises this term "contracture" therefore, as applied to the state of the muscle in peripheral neuritis, and prefers to call the state one of fibro-tendinous contraction. He, however, recognizes, more fully than do most writers, the early painful cramps of multiple neuritis which occur before the muscular degeneration is complete; and these he calls by their proper term, "cramps." This distinction of Babinski may be a just one, and yet it appears somewhat artificial, when we consider that these persistent cramp-like states, especially in the legs, in the early stages of multiple neuritis, pass by imperceptible gradations into the more permanent contractures of the later stages. I would prefer to preserve the term "cramp" for the fugitive symptoms sometimes seen in the early stages of the disease, which I have already referred to in a special section, and to regard the more permanent, painful, cramp-like contractures of the later stage, passing into permanent contractures, as a distinct process in some way associated with the process of degeneration, and especially marked in the least paralyzed and least opposed muscles.

In connection with motor disorders may be considered disorders of the reflexes. As a rule, the tendon reflexes are very promptly affected in multiple neuritis. They are, almost without exception, lessened or abolished. Some observers have claimed that they have seen an exaggeration of the deep reflexes in peripheral neuritis. Strümpell, Möbius, and Babinski are among those who make this claim. I have never seen this exaggeration and believe it must be a very rare phenomenon. While there may be nothing in theory absolutely to oppose the idea of this exaggeration, yet practically we know that the phenomenon rarely occurs. It is possible, for instance, to suppose that in the initial stage of a neuritis, due to some poison acting upon the nerve endings—especially upon the sensory nerve endings—the excitability of the neuron might be temporarily increased, and that under these circumstances the reflexes might be exaggerated. Déjerine has noted that the reflexes may persist in some cases; and it is true, in my observation, that cases occur in which they are not absolutely lost for a long period. In the rare instances in which there is an initial exaggeration of the reflexes, this phenomenon is soon replaced by a gradual diminution and final disappearance of this sign. All the tendon reflexes may be affected or abolished in multiple neuritis. Those most conspicuously involved, however, are the knee-jerks. In cases in which they are not promptly abolished, they may present a decreased and rather more sluggish contraction than is normal. This alteration in the character of the jerk may be in some cases as important a pathognomonic sign as the diminution itself.

The superficial or skin reflexes are not quite so uniformly diminished and abolished as are the deep. In the early stages, in which hyperæsthesia of the skin is a well-marked symptom, these skin reflexes may even be exaggerated. This exaggeration no doubt depends upon irritation of the endings of the sensory nerves. In severe cases, in which anæsthesia and motor paralysis are well marked, the skin reflexes are almost uniformly abolished. A very sluggish cremasteric reflex may, however, persist in even severe cases, although this is rare. Among other variations that may be noted in the reflexes is the variation in individual reflexes as compared with others. It does not follow, for instance, that all the reflexes, either deep or superficial, must be abolished at the same time. Thus, one reflex may persist much longer than another, and some superficial reflexes may even appear exaggerated after the other reflexes, especially the deep, are abolished. The deep reflexes may be abolished, for instance, in the legs and not be affected in the arms. These differences of one reflex from another are due simply to variations in the location

of the disease process. They simply indicate that the neuritis is more marked in one set of nerves or one region of the body than in others.

The next important symptoms connected with the motor phenomena of multiple neuritis are the changes in the electrical reactions of the paralyzed muscles. Such changes are uniformly seen in all varieties and types of polyneuritis. They may vary not a little, however, in character and degree, according to the intensity and the location of the disease process. In order to understand these variations it is, of course, necessary to understand the normal reaction of nerve and muscle to electricity. These have already been described in the section on electrotonus (p. 21), to which reference can be made.

The earliest change in the electrical response of the muscle in multiple neuritis is to the faradic current. The faradic irritability is rapidly diminished in most cases and is soon completely abolished. This takes place even before there is well-marked degeneration and atrophy of the muscle; in fact, it may occur in a few days. It depends, as already explained, upon the physiological fact that muscular tissue, when deprived of nerve influence, will not react to faradism. Consequently this loss of faradic excitability is a sign of a change in the nerve rather than in the muscle. It indicates clearly that the axis cylinder is no longer able to transmit impulses. It is for this reason that this loss is, as a rule, the first change in electrical irritability to show itself, since it does not depend upon advanced degenerative changes in the muscular tissue, but simply upon a lesion that acts very promptly to cut off the peripheral motor neuron. It is thus easy to understand also why variations occur in the promptness and completeness of this loss of faradic contractility. In cases in which the poison or destructive agent acts slowly and insidiously, the faradic contractility may not be abolished at once. This is for the simple reason, of course, that some of the normal nerve impulses continue to flow. This preservation of a somewhat weakened response to faradism may be seen even in cases in which the motor paralysis is complete, and indicates that the motor neurons, though so much paralyzed as not to be able to transmit motor impulses, may still maintain some nutritive control over the muscle. The time required for the complete abolition of faradic contractility varies, of course, in different cases. A complete and sudden destruction of the axis cylinders, as by section for example, is followed by diminution and loss of faradic contractility in a few days; in fact, the diminution may begin in a few hours. Few cases, however, of ordinary

multiple neuritis present such an abrupt onset dependent upon such a distinct lesion. Consequently the loss to faradism does not usually occur bruskly. In cases of inflammation of the facial nerve, however, the decline and loss of faradic contractility are very early phenomena. In alcoholic multiple neuritis, while not quite so prompt, they are yet, as a rule, not long delayed. This abolition of faradic contractility may vary very much in different regions of the body in the same patient. Thus faradic contractility may be well preserved in some muscles and much diminished or totally abolished in others. This fact demands care in observation, for erroneous conclusions may be drawn from a hasty and incomplete examination. Even the muscles in the same segment of the limb will vary among themselves. Thus the extensor group of the forearm and leg may show complete abolition of contractility, while the flexor muscles will still give a response. In some muscles, even quite paralyzed and markedly atrophied, there may occasionally be found a slight faradic contractility. This is probably due to the fact that all the fibres in the muscles are not equally degenerated, because all the nerve fibres supplying them are not equally inflamed.

As the faradic contractility declines, changes much more complex occur in the galvanic excitability of the muscle. These changes, unlike those to faradism, are indicative of the reaction of the muscle itself and not of the nerve fibre. They depend, in fact, upon the cessation of the influence of the nerve fibre upon the muscle fibre and the gradual degeneration of the latter. Hence they are properly to be regarded as muscular phenomena, and they are, properly speaking, the true reactions of degeneration. First, in order of time, is a slight initial increase in the galvanic excitability. This is shown by the response of the muscle to a milder current than would normally excite a contraction. This initial increase is probably a sign of irritation. It is but brief, lasting at best, as a rule, but a few days. It is rather more common in abrupt, rapidly acting lesions than in those that ordinarily cause polyneuritis. Thus it is much more likely to be seen after section of the nerve or after such an inflammation as occurs in the case of the facial nerve after exposure to cold, than it is in alcoholic multiple neuritis. In fact, its occurrence at all in alcoholic multiple neuritis is perhaps somewhat theoretical. I do not recall clearly having seen it in any instance. After a few days, the rule is for the galvanic excitability of the muscle to diminish slowly but progressively. It is not abolished rapidly like the faradic contractility. These alterations—*i.e.*, the increase and decrease in galvanic irritability—are called *quantitative* changes. This progressive diminution is almost constantly seen in polyneuritis. Its rapid-

ity and intensity will, of course, depend upon the severity of the case. In some cases, or in some muscles in some cases, it may not advance to complete disappearance. It may even escape observation in mild cases, unless care is used to estimate the strength of the current and to compare the reaction of the diseased muscle with that of a healthy one.

As the quantitative changes take place, changes also occur in the order of the reaction of the current to the two poles. As will be recalled by reference to the section on normal electrotonus, a muscle when innervated by a healthy nerve responds first and most vigorously to the cathodal or negative pole at the closure of the current; next to the anodal closure or the anodal opening—these two, in fact, varying slightly. But, as a rule, the response of the muscle to the anodal closure is more marked than to the anodal opening. Finally the muscle responds least of all to the cathodal pole at the opening of the current. Consequently the normal formula, as already given, is as follows:

$$CCC > ACC > AOC > COC$$

or as occasionally happens:

$$CCC > ACC = AOC > COC$$

As the muscle begins to degenerate, a change occurs in the order of its reaction as expressed by the normal formula. First, the response to the cathode or negative pole at closure diminishes—*i.e.*, it requires a stronger current to elicit this response. At the same time the excitability of the muscle to the anode or positive pole at closure, while diminishing slightly, does not do so in proportion to the cathode. Hence in time the ACC may equal or even exceed the CCC. Further, the response of the muscle to the anode at the opening of the pole, never as a rule very conspicuous, diminishes and disappears entirely. Finally, the response of the muscle to the cathode or negative pole at the opening of the current, which is practically never seen in the normal muscle except with an exceedingly strong and almost unbearable current, may now appear, especially in advanced cases of degeneration. As a consequence, the typical and complete reaction of degeneration is expressed in the following formula:

$$ACC > CCC > COC > AOC$$

the last element being practically abolished.

These alterations constitute what are called the *serial* reactions of degeneration.

In peripheral multiple neuritis these serial changes may present many variations, according to the severity and extent of the lesion. In the early stages or in mild cases there may be simply a diminu-

tion in the CCC so that it becomes about equal to the ACC and the response of the muscle to either pole at the opening of the current is not seen. This modified form of reaction usually accompanies a slight diminution, or even in some cases a complete loss, of faradic contractility. In more severe or more advanced cases, the CCC may fall below the ACC, while the AOC is entirely absent and the COC has not yet manifested itself. In my observation, this is the most common alteration of the electrical reaction in multiple neuritis and may be expressed thus:

ACC > CCC (AOC and COC not appearing.)

The appearance of the COC even in advanced cases of multiple neuritis is not, in my observation, a common occurrence. The formula of degeneration as thus given is maintained, as a rule, during the continuance of the stage of degeneration. The ACC continues to maintain its precedence, although with gradually diminishing force. In cases in which the muscle proceeds to ultimate complete degeneration, the CCC gradually diminishes and is ultimately lost. The last response of the expiring muscle is to the anodal pole at the closure of the current, the ACC.

The changes thus described may be taken as a type of the reactions of degeneration as occurring in ordinary multiple neuritis. Variations from this type undoubtedly occur, but they are all in line with the departure from the classical formula of normal reactions here given. It occasionally happens that these serial reactions of degeneration are not so well marked as the degree of motor paralysis would lead the observer to suspect. This may be due to the fact, already explained, that in mild cases the motor neurons may be sufficiently incapacitated not to be able to transmit motor impulses, but may still be able to maintain some nutritional control over the muscle. In mild cases, for instance, there may be merely a slight diminution in the faradic contractility without any alteration in the normal formula for galvanic contractility.

A third change in the response of the degenerating muscle to galvanism is seen in its manner of reaction. Instead of the quick, tonic contraction so characteristic of the response of a normal muscle to electricity, there may be a slow or sluggish response which is unmistakable. This usually occurs only when the other electrical phenomena of degeneration are well marked. It is called the *modal* change.

Finally, in some cases of neuritis the muscle may present the phenomenon known as "duration tetany." This consists in a prolong or sustained contraction of the muscle during the continuance of the passage of the current. It is in marked contrast to what oc-

curs in health, when, as is well known, the muscle, as a rule, contracts only during either the making or the breaking of the current—*i.e.*, during the instant of the rapid change from one state of electrotonus to another.

The Sensory Symptoms.—Sensation is usually profoundly affected in every case of multiple neuritis. It may be affected in one, or several, or all of its modes; and it may either be excited or paralyzed.

One of the most common and characteristic of the affections of sensation is *pain*. This is usually an early phenomenon. It is marked especially in the muscular masses themselves, but is also experienced in the nerve trunks. The character of the pain varies slightly. In some few cases it is described as sharp and lancinating, but for the majority of cases it is burning and exquisitely severe. In the early stages of the disease, in fact, it is the most urgent symptom, and one which at this time may be readily mistaken. Thus, for instance, it has not infrequently been attributed to rheumatism. Its favorite seats are in the paralyzed muscular masses and in the overlying skin. So exquisitely tender and sensitive do these parts become that the patients cannot bear the slightest touch or manipulation. Even contact with the bedclothes may be unbearable. This was noted more than a hundred years ago by Lettsom, who said that the tenderness of the parts was so exquisite that the weight of the finger excited shrieks and moaning. In consequence, the patient views with evident apprehension any advance towards examining his legs and arms. Handling the parts or even gently stroking the skin may cause cries of pain, but the greatest agony is experienced from firm pressure on the nerve trunks at their more exposed points, and especially from squeezing the paralyzed muscles. This tenderness of the muscular masses is so characteristic and constant that it may be said to be pathognomonic of the disease, and is seen in no other affection. The soles of the feet, too, are often exquisitely sensitive. Even in alcoholic patients with marked hebetude or stupor, this symptom can be elicited usually without difficulty. Pains of a neuralgic character are also spontaneous in this disease. They occur especially when the patient attempts any voluntary movement, and are then usually associated with the cramp-like condition already described. In attempting to move the leg the patient, if not too badly paralyzed in the arms, may endeavor to assist the movement by slipping his or her hand under the thighs, as I have lately seen a patient do. The expression of pain, or even terror, on the patient's face is sometimes striking, and it is not uncommon for women to shed tears because of their suffering. These pains are usually worse

in the legs, feet, toes, and about the knees, although they are not wanting in the upper extremities.

Parœsthesia is very commonly seen in multiple neuritis. This may consist in sensations of tingling, formication, etc. These sensations are variously described by the patients themselves. Thus one patient described them as a sense of cold water trickling down the leg. For another patient they were like the contact of some hot substance. A sense of combined tingling and numbness, however, is perhaps the most common of these paræsthetic symptoms.

Anæsthesia is an almost constant symptom of multiple neuritis. It varies very much, however, in both location and extent, according to the circumstances of the case and the cause of the disease. In alcoholic cases it is almost universally found in some degree at some stage or other of the affection, but in lead cases it is not so common. It may be distributed somewhat irregularly, and is more constantly seen in the legs below the knee than elsewhere. It is a very constant symptom in the ataxic type of the disease, and is then no doubt the cause of the incoördination seen in these cases. The modes of sensation most involved are the tactile and thermal. The former is especially impaired, but in many cases it will be found that the patient cannot readily distinguish heat and cold. Analgesia, however, is not usually seen. On the contrary, as already described, there is hyperalgesia even to the slightest touch. This, however, is not always so in the advanced stages of severe forms of multiple neuritis, although even in them I have seen hyperalgesia well marked. The combination of anæsthesia with some hyperæsthesia or hyperalgesia is a striking characteristic of the disease in some cases. The alteration of the thermal sense is not always striking, and may even be absent in cases in which anæsthesia, especially to a slight touch, is well marked. This characteristic of the anæsthesia should not be ignored. Thus, while a slight touch may not be perceived, deeper pressure may be felt both as a tactile and as a painful sensation. Retardation of sensation may also be noted in some cases. Some authors claim that thermal anæsthesia is not common in multiple neuritis, yet I have seen it in severe cases in which the anæsthesia also was well marked. It may be said in a general way, therefore, that a slight degree of tactile anæsthesia is the first and commonest form observed, but this may coexist with extreme sensitiveness to pain and with preservation of the thermal sense. Later, tactile anæsthesia may be more profound and is then associated with some degree of thermal anæsthesia, but the sensitiveness to pain, especially on deep pressure, is rarely abolished even in advanced stages of the most severe cases. Sensitiveness to the electrical current may be

impaired if a very mild current is applied lightly to the skin; but stronger currents, especially if they cause muscular contraction, are very painful in the early stages of the disease. Later, as the patient tends to recover, the electric current, both faradic and galvanic, can be borne without much discomfort.

The Trophic Lesions.—The trophic lesions in multiple neuritis are varied. The most constant and striking is atrophy of the muscles. This, as already explained in a preceding part of this paper, is an inevitable sequence of degeneration or destruction of the peripheral motor neurons or their axis cylinders from any cause whatsoever. Thus it occurs after injuries to nerves as well as with inflammation of them, and the process, so far as the muscle is concerned, is identical in both of these instances. In multiple neuritis, of course, this degeneration depends upon the destruction or obstruction of the axis cylinder in or near its peripheral distribution. The muscular fibre is then cut off from its true trophic centre, which is in the cell body of the motor neuron in the anterior horn of the gray matter of the spinal cord. This process of degeneration in the muscle begins promptly when the axis cylinder is interrupted or destroyed by inflammation. In peripheral multiple neuritis it is usually an early symptom. The paralyzed and painful muscles become soft and flabby. The natural muscular tone is abolished and the muscle loses rapidly, as a rule, in bulk. As, however, the degree of severity and completeness of inflammation of the nerve trunk varies in different cases, so this phenomenon of atrophy will vary. In mild cases, while some degree of it is always apparent, it may not advance to complete destruction of the muscle. In severe cases, however, of long standing, the atrophy may be so complete that apparently but little if any true muscular tissue remains. This is seen especially in the peroneal muscles in the leg and in the extensor muscles of the wrist and hand. This atrophy of the muscles is never, as a rule, associated in multiple neuritis with fibrillation, as is usually seen in cases of anterior poliomyelitis. Exceptions to this rule are stated by some observers to occur, but they must be very rare. The paralysis always precedes the atrophy. In some rare cases, in fact, there may be a marked paralysis without very distinct amyotrophy. This probably indicates that the axis cylinder has not been entirely destroyed but only rendered incapable of transmitting motor impulses, but that it may still preserve its continuity sufficiently to maintain its trophic influence. It is thus seen that the two phenomena, paralysis and atrophy, are not necessarily coexistent, although in the vast majority of cases the one follows rapidly upon the other. This dissociation is still further seen in the decline of the disorder. As the patient tends

to recover, the atrophied muscles begin to respond to the volitional impulses, and may even regain considerable activity before the nutrition begins apparently to improve. This, however, may be only in appearance and not in reality, because the activity of the muscle being much more readily appreciated by the eye, may seem to advance more rapidly than its regeneration. It is probable, in fact, that in a great majority of cases, at least, the two functions proceed together, and that as the muscle regains its power to contract it is also beginning to reëstablish its nutrition. This is scarcely the place to enter into an exhaustive discussion of the question whether any types of progessive muscular atrophy may be dependent upon a progressive peripheral neuritis. Charcot and Marie have presented a group of cases of progressive muscular atrophy in which, in the absence of autopsies, they proposed the hypothesis that the process was dependent upon a peripheral neuritis. Hoffmann has likewise recorded such cases, for which he proposes the name of "progressive neurotic muscular atrophy." He thinks that these cases hold a middle position between those that are caused by a degenerative process in the spinal cord on the one hand, and the true primitive myopathies on the other. This type of case, if it is truly existent, must depend upon an extremely slow and progressive neuritis which invades the peripheral nerve ending, not all at one time, but in series or groups. Consequently, in such cases the muscular atrophy, attacking only fibre after fibre of the muscle, would appear to go before the paralysis. This is because the muscular fibres still intact would retain a power of contraction. This type of case, however, can scarcely be included in a description of peripheral multiple neuritis such as is designed here. The process resembles one of progressive degeneration rather than of acute neuritis, widespread and coming on rapidly, such as we are here considering. It is worth while to recall, however, that this type of slow progressive muscular atrophy, possibly due to a peripheral neuritis, has been observed to follow the infectious diseases, such as typhoid fever and measles, as has been described in the section on etiology.

Œdema or swelling of the paralyzed members is seen in some cases of peripheral neuritis. It is especially common in beriberi. It is also seen to advantage in some cases of alcoholic neuritis. It is especially prominent in the legs below the knee, although it is not unusually seen also in the forearms and hands. In estimating the true significance of this symptom, it is well to recall that, in alcoholic cases especially, the heart and lungs may be seriously affected and that the œdema may be promoted by impairment of these organs. Still, we know that œdema can and does occur as a result of nerve

injury and subsequent inflammation, and therefore there is no reason for hesitating to regard this symptom as in the nature of a trophic or a least a vasomotor affection. Swelling and even effusion into the joints have been recorded by some observers. Deformities of the joints, in fact, in long-standing cases are not uncommon. These are no doubt promoted by the contractures and disuse, but are also evidently partly trophic in character. The finger-joints especially may become swollen and tender so as to simulate rather closely a rheumatic or gouty affection. Glossy skin is usually seen in association with the œdema. It may even be present, in fact, when very little, if any, swelling of the parts is present.

In long-standing cases the nails and hair may be affected, just as is seen and has already been described in traumatic neuritis.

Destructive trophic lesions of the skin, such as *herpes*, *pemphigus*, and *whitlow*, are not seen in the commoner types of multiple neuritis. Thus in alcoholic cases I do not recall ever to have seen them. They are very common, however, especially destructive whitlows, in the anæsthetic form of leprosy, which depends upon a neuritis. The reason for the absence of these destructive skin lesions in alcoholic multiple neuritis is not easy to state. The affection of the nerves has all the characteristics of an irritative and destructive process, and yet the clinical fact remains that we do not have these lesions in cases due to alcohol and lead. The same may be said of bedsores. They are not common in multiple neuritis. Their absence on the trunk may possibly be due to the fact that the nerves of the trunk are not, as a rule, so seriously involved as the nerves of the extremities. Consequently the nutrition of the trunk does not suffer so much as does that of the arms and legs. Bedsores may form, however, on the heels and ankles and also about the hips in alcoholic multiple neuritis if care be not taken to guard against them. In a case of extensive multiple neuritis, following typhoid fever, which occurred in my service in the Methodist Hospital, very obstinate bedsores occurred in the region of the malleoli. One of these continued as an open indolent ulcer for many weeks, in spite of the fact that pressure was carefully avoided and that the sore was dressed with antiseptic precautions. Probably one reason why bedsores do not form more frequently on the heels and ankles and hips in multiple neuritis is that these parts are accessible and hence can be guarded from such results.

Visceral and Internal Disorders.—In many cases of multiple neuritis there are symptoms referable to the viscera and other internal parts and organs. These may be divided into two classes; first, those that are caused directly by the poison which causes the neu-

ritis, and second, those that are due directly to the neuritis itself. The first class cannot be appropriately considered in detail here. They are the symptoms merely of a general systemic affection and are not necessarily associated with neuritis; in fact, they frequently occur in cases in which there is no neuritis. Still, as forming a part of the clinical picture, they may, at least, be briefly referred to. In alcoholic cases, for instance, it is not unusual to find the symptoms of gastric, hepatic, and renal disease. These are such as may occur in any case of chronic alcoholism. Occasionally a patient presents the symptoms of a gastric catarrh. There is irritability of the stomach, with indisposition to take food, and very rarely nausea and vomiting. These symptoms, however, are, in my observation, very uncommon. In cases in which they do occur the forced abstinence from alcoholic drink soon establishes a cure, and henceforth the patient may be able to take a fair quantity of nourishment. I have noticed in several of these patients a jaundiced hue of the conjunctiva, with some enlargement and tenderness of the liver. These symptoms probably indicate a chronic catarrhal hepatitis. I have never yet seen a case of cirrhosis of the liver complicating multiple neuritis. It seems to me that for some reason alcoholic neuritis does not occur readily in the type of patients who develop the cirrhotic liver. This liver occurs especially in men who have been, as a rule, high livers. They are often stout, well-fed individuals, who have not been exposed to great privation and over-exertion in cold and wet places. On the other hand, multiple neuritis, as we know, is very likely to occur, especially among women, in individuals who have been half starved and suffered much exposure. It may possibly be a logical deduction from these facts that the peripheral nervous system suffers more readily in these individuals than does the liver. I recall, however, one instance of a young woman, aged twenty-eight, the subject of chronic alcoholism, who had suffered much privation and exposure, and who died of cirrhosis of the liver. In this instance, while there were marked emaciation and apparent atrophy of the muscles of the legs and arms, there had not been during life any well-marked symptoms of multiple neuritis. Diseases of the kidneys are not unusual in old alcoholic cases. I think, however, that, as a rule, the symptoms of advanced kidney disease are not very common in cases of alcoholic multiple neuritis.

Disorder of the heart is very common in some forms of peripheral neuritis. In the alcoholic type, the derangement of the heart constitutes one of the gravest complications. This is manifested by extreme rapidity and weakness of the heart's action. It is not uncommon in these cases to have a pulse ranging constantly above 100 and

it may even be as high as 120, 130, or 140. I have seen several cases in which the latter rate was maintained for a number of days. In one case, in a woman in the Philadelphia Hospital, this weakness of the heart was the cause of sudden and unexpected death. It may be worth while to state that the sudden death in this case seemed to be due to the application over the heart of an ice-bag, which was made at the suggestion of an eminent clinician who saw the case in consultation. This tachycardia is generally considered by authors to be due to involvement of the pneumogastric nerve. Déjerine, quoted by Babinski, has noted alterations in this nerve in a case of peripheral neuritis in which tachycardia was a symptom. It is easy to understand, of course, how inflammation of the pneumogastric nerve may cause tachycardia and even sudden death. The inhibitory action of this nerve upon the heart is well understood. When for any reason this is interfered with, as by inflammation, grave disorder in the rate and rhythm of the heart inevitably results. In cases of intermittent paralysis of the cardiac branches of the vagus, according to Landois and Stirling, acceleration of the pulse above 160, 200, and even 240 beats a minute has been noted. At the same time, the beats vary much in rhythm and force. In this connection, also, it is well to recall that if the trunk of the vagus be paralyzed there are labored, deep, and slow respirations. In diphtheritic paralysis, even more than in alcoholic paralysis, fatal impairment of the nervous mechanism of the heart is to be feared. It is not an uncommon cause of sudden death in these cases.

The tongue, soft palate, and pharynx may be involved in multiple neuritis. This is not commonly seen, however, in alcoholic cases. In diphtheritic cases it is far from uncommon. The veil of the palate and the mucous membrane of the pharynx may be quite anæsthetic, so that the reflex movements, normal in these parts, on tickling or irritating with a sharp instrument, may be entirely abolished. Paralysis of the velum palati in diphtheritic cases is the cause of some characteristic symptoms, such as the nasal speech, slow and difficult articulation, and snoring during sleep. Disorders of deglutition are observed, especially on attempts at swallowing liquids. These may regurgitate through the nose. Solid or semi-solid substances are swallowed more easily, as they, of course, do not find a ready passage through the posterior nares. This difficulty in deglutition is much increased, of course, if the muscles of the pharynx, as sometimes occurs, are paralyzed. Then even solid substances may be swallowed only with great effort and the risk to the patient is much increased from the difficulty in administering food. One of the risks in these cases is from the passage into the larynx of por-

tions of food. These may not be perceptible to the patient because of the anæsthesia of the parts and the consequent abolition of the normal reflexes. Paralysis of the tongue is not so common in these cases. Babinski notes that the paralysis may even extend, in some cases, to the lips and cheeks. Kast has noted, in one case of polyneuritis, not only paralysis, but atrophy of the tongue.

Disorders of digestion have already been noted. Reference may be made here, however, to the question which has been raised by some observers whether painful gastric crises, similar to those observed in locomotor ataxia, may occur in polyneuritis. Confusion may arise here on account of the painful state of the stomach due to a gastric catarrh, especially in alcoholic cases. Such a gastric catarrh, as already said, may be present in these cases, but it cannot be said to be a true trophic or neurotic affection, being due rather to the direct irritating effect of alcohol upon that viscus. Hence it seems to me erroneous to regard it as analogous to the gastric crises of locomotor ataxia. Considering, however, the irritation and subsequent paralysis of the pneumogastric nerve, it may be considered rather remarkable that gastric symptoms due to this cause are not more commonly observed in multiple neuritis. Thus we might suppose that nausea and vomiting would be more common symptoms. I have observed nothing, however, to establish such an opinion.

Disorder of the intestines is not common in any form of multiple neuritis, if we except the well-known colic of lead poisoning. This symptom, however, cannot positively be claimed to be due to an affection of the nerves. It has been suggested as a mere hypothesis that this lead colic is due to the action of the poison upon the nerves of the intestine, but this has not been established as a fact. Constipation is frequently noted in peripheral neuritis, but that it is due to a paralysis of the walls of the intestines is not proven. Paralysis of the rectum and the sphincter muscle of the anus is a very unusual symptom in multiple neuritis. In cases in which incontinence of fæces occurs, this is usually due rather to the hebetude or stuporous condition of the patient than to a true local affection at the terminus of the gut.

The Special Senses.—The special senses, with the exception of the eye and its muscular apparatus, are seldom if ever affected in any form of multiple neuritis. I have never seen any affection of smell, taste, or hearing in this disease. It is possible that in some alcoholic cases which I have seen the senses of smell and taste may have been somewhat blunted, but owing to the mental torpor and confusion of these patients the tests for these senses have seldom been satisfactory.

I have made no observation, nor have I seen reports from others, of any affection of the auditory nerve.

The optic nerve and some of the muscles, both extrinsic and intrinsic, of the eye, may be profoundly affected in some forms of multiple neuritis. These affections are much more common in the neuritis due to lead and to diphtheria than in that caused by alcohol. In the latter forms any affection of either the nerve or the muscles is rare, although instances have been reported. It is possible that sufficiently careful examinations of the optic nerves and the optic muscles are not made in cases of alcoholic multiple neuritis, and that affections of the kind here noted would be more commonly reported if such examinations were made.

Paralysis of the extrinsic muscles of the eye is not unusual in diphtheritic paralysis. The most common is paralysis of the external rectus, sometimes in both eyes, causing a convergent strabismus. This is not uncommonly seen in children suffering with this disease and may be one of the earliest symptoms noted by the parents or friends. Thus a mother reported to me that the first thing she noticed in her child, who had been convalescent several weeks from diphtheria, was that he squinted. This paralysis causes diplopia and the patient will probably complain early in the disease of double vision. Paralysis of the third or oculomotor nerve is not so common in diphtheria, although instances of it are on record. Thus, in a noteworthy case, Remak observed an associated paralysis of the oculomotor and abducens nerves in each eye; and at the autopsy Mendel found changes in the roots of these two nerves (quoted by Babinski). In some of these grave forms of complete external ophthalmoplegia the diphtheritic poison attacks the oculomotor nuclei in the midbrain beneath the aqueduct of Sylvius and the associated nucleus of the sixth nerve in the pons. These cases, then, constitute examples of the superior polioencephalitis of Wernicke. In alcoholic cases paralysis of the extrinsic or intrinsic muscles of the eye must be extremely rare. Ptosis and paralysis of the external rectus muscle has been noted in a very few cases. Babinski quotes Thomsen as having observed paralysis of the external rectus four times and ptosis twice in twenty-six cases of alcoholic neuritis. This is certainly an exceptional experience, as probably few observers could claim that they had seen paralysis of the ocular muscles in twenty-five per cent. of their cases of multiple neuritis.

Paralysis of the ciliary muscle causing loss of power of accommodation is a very common sequel of diphtheria. It is probably absent in few, if any, cases of diphtheritic paralysis. It is the cause of the weakness of vision complained of by these patients even in the

absence of other marked paralytic symptoms. Adults are rather more likely to notice and speak of paralysis of the muscle of accommodation than are children, especially small children. It interferes with reading and with the use of the eyes for other fine work, and consequently is quickly noted by older children and by adults. It is not usually associated with any loss of power of convergence of the eyes, and as a rule, to which there are probably few exceptions, there is no paralysis of the iris. Consequently the pupil contracts freely to light. This is not to be confused with the well-known Argyll-Robertson pupil, in which the pupillary reflex to light is lost, but its reaction on accommodation remains. This paralysis of accommodation from diphtheria is bilateral, and it is often one of the most persistent of the phenomena of postdiphtheritic paralysis. It is not a symptom observed in any other form of multiple neuritis, so far as I know.

Paralysis of the iris is a rare symptom in any form of multiple neuritis—so rare, indeed, as to constitute of such cases a sort of clinical curiosity. The Argyll-Robertson pupil is not seen in any form of multiple neuritis. This pupil, in which the reflex to light is lost while its reaction on accommodation is preserved, is generally held to depend on an affection of the nerve centres, and is seen especially in locomotor ataxia and more rarely in general paresis. Its appearance in any case should throw a doubt upon the accuracy of the diagnosis of multiple neuritis and should raise a suspicion of a degenerative process in the nerve centres. Thus in the cases noted by Eperon (quoted by Babinski[186]), in which this author noted tabetiform symptoms associated with toxic amblyopia from tobacco or alcohol, the Argyll-Robertson pupil was associated with fulgurant pains and abolition of the knee-jerks; and the suspicion arises that the patients were suffering with posterior sclerosis.

The optic nerve may be inflamed or degenerated as a result of the systemic intoxication which sometimes causes polyneuritis. In chronic lead poisoning, optic neuritis is not uncommonly seen. Retrobulbar neuritis may also occur in cases of chronic poisoning from alcohol, tobacco, quinine, and some other toxic substances. This symptom, however, is not necessarily or even commonly associated with multiple neuritis. The resulting symptom is an amblyopia caused usually by a central scotoma. The periphery of the field may retain its integrity, and many of these patients are only conscious of a slight impairment of vision. It is said that they see better in the dusk or in a dim light. In diphtheritic paralysis involvement of the optic nerve is not common (De Schweinitz[346]).

Mental Symptoms.—In cases of alcoholic multiple neuritis mental

symptoms are not uncommon. As Clouston has pointed out, a long-continued soaking in alcohol is damaging to the brain in its mental, motor, and trophic functions. The type of mental disorder seen usually with multiple neuritis is a low grade of stupor or confusional insanity. The patient's mental faculties all seem to be blunted. In some cases it is difficult to fix his attention or to obtain from the patient himself a clear history of his disease. The memory is very much impaired, especially for recent events. There may be a mild form of delirium, but not quite so well marked or active as in the well-known delirium tremens. In this mental state unsystematized delusional ideas are not infrequently seen, and especially confusion of personal identity and of time and place. Occasionally, in more chronic cases, the delusion may become fixed and more systematized and may persevere for some time even after the patient's mental faculties seem to be well restored. These fixed delusions are usually of suspicion and fear. The confusions of personal identity are perhaps among the most characteristic of these mental symptoms. The patient fails to recognize or properly place his friends and kindred, and the confusion may even extend, in some cases, to his own personal identity. Hallucinations of sight and hearing are not common in multiple neuritis. Of the two, the latter are the more frequently seen. Besides alcohol, the poisons of some of the infectious diseases, as typhoid and typhus fevers, tuberculosis and puerperal sepsis, can cause a psychosis which is practically identical with that induced by alcohol. One peculiarity of this psychosis is the tendency for it to be worse at night, especially in the early hours of the night. Similarly, and probably for the same reason, the delirious state is intensified if the patient is left entirely to himself. The reasons for this are apparently because, the attention being then unattracted by external objects, the patient's mind lapses into a dream-like state. Lasègue, noting the resemblances of subacute alcoholic insanity to dream-like states and reveries, said that it was not an insanity, but a dream. Regis[11] calls attention to the fact that this psychosis is always of a melancholic type.

The loss of memory is a striking symptom in the insanity of alcoholic neuritis. It is, in fact, a fundamental symptom upon which probably many of the other symptoms depend. It is most marked for recent events, especially for all events that have occurred since the beginning of the patient's malady. The memory for remote events is not nearly so much impaired, although, as the patient's attention is difficult to fix, this fact cannot always be satisfactorily tested. The confusion of events and of personal identities may be explained, first, by the weakness of perception and, second, by the

loss of memory. Percepts are not accurately formed or clearly registered; hence confusion results in the patient's mind. Not infrequently amnesia is almost the only manifestation of mental disorder in cases of multiple neuritis. Korsakoff [110] calls attention especially to the fact that the patient under these circumstances may even give the impression that he has the use of all his faculties.

From the clinical standpoint it may be said that the type of psychosis seen in multiple neuritis from any cause is confusional insanity. This type, as is well known, may occur after various infectious diseases, such as typhoid fever and puerperal sepsis, without the complication of a peripheral neuritis. It simply indicates that the neurons of the cerebral cortex are poisoned; and that in some cases this poison has, in others it has not, involved the peripheral motor and sensory neurons as well.

The prognosis in these cases is in the main good so far as the mental symptoms are concerned. From my observation I should say that in severe cases the prognosis for the mental symptoms is better than for the paralysis. Thus I have seen patients in whom the mental faculties were gradually restored long before the peripheral symptoms had disappeared. Cases may happen, however, especially when the exposure to alcohol has been long continued, in which the mental disorder may advance to a chronic insanity with fixed delusions of persecution and with a tendency to mental deterioration or dementia.

Evolution.

Multiple neuritis may present itself as a disease either of abrupt onset and acute and rapid course, or of slow and insidious onset with a protracted and chronic course.

In the first type of cases, the symptoms may all be established in the course of a few days. There may even be slight initial rigors, with some febrile reaction. In alcoholic cases especially pain is a conspicuous symptom, but loss of power rapidly supervenes and tachycardia and mental symptoms complete the picture. In these cases the patient is bedridden from the very beginning and presents the appearance of grave illness. Such cases not infrequently prove fatal. They are occasionally seen also following diphtheria, although in this instance pain is not marked. Very rarely lead and arsenical poisoning, the latter especially, present this type of abrupt onset and acute course.

In the second class of cases, the onset is slower, and some days, or even weeks, may elapse before all the symptoms are manifested. In these cases, also, pain is an early and urgent symptom, and the

patient may even be suspected of suffering with rheumatism or neuralgia. Motor symptoms, however, manifest themselves in time and eventually the patient may be bedridden. Some of these cases are mild and the patient may even not be totally incapacitated. They usually seek their beds, however, from choice, as the pains are much relieved by absolute rest. Between these two extremes all grades of severity are noted. In diphtheritic cases, as already said, the symptoms of paralysis may not manifest themselves until the patient is well advanced in convalescence from the primary disease. The further advanced he is, as a rule, the milder the symptoms will be. Exceptions to this rule, however, occur. I have seen in one instance a widespread diphtheritic paralysis occur in a child as late as the fourth week of the primary disease. In typhoid fever the symptoms usually manifest themselves before the patient leaves his bed. Pain and tenderness of the nerve trunks are the earliest of these symptoms. They occur, in fact, in some cases of typhoid fever in which motor paralysis never becomes conspicuous. Such cases are usually of short duration and of favorable termination. In tuberculous patients, the evolution of peripheral neuritis may be very slow and insidious. The disease, in fact, is probably not infrequently overlooked in these cases, being disguised by the general prostration and failure of the patient, owing to the pulmonary disease.

Clinical Forms.

As already said, multiple neuritis presents itself under a variety of forms, according especially to its exciting cause. The chief of these will be briefly indicated here in order to complete the clinical picture of the disease.

Alcoholic Polyneuritis.—The description already given in detail of the symptoms of multiple neuritis refers so directly to the alcoholic type of cases that little more need be done here than briefly to group symptoms and to recapitulate. In alcoholic multiple neuritis the salient features are the acute pains in the inflamed nerves and especially in the muscular masses and in the skin overlying them; the motor paralysis, involving especially the extensor muscles, with consequent hand- and foot-drop; contractures; muscular atrophy, and the appearance of the reactions of degeneration. Severe cases are complicated with tachycardia and paralysis of some of the muscles of respiration. Finally, there is often some grade of a confusional psychosis.

The onset in these cases is, as a rule, insidious; although cases occur in which the *début* is brusk, especially after exposure to cold

and wet, and occasionally after the puerperium. The onset of the disease is marked usually by the pains, burning or neuralgic in character, especially in the extremities and more particularly about the knees, legs, ankles, toes, and soles. These may simulate rheumatism or even locomotor ataxia, and have led in numerous cases to mistakes in diagnosis. Later the paralysis manifests itself and soon disables the patient to such an extent that he becomes bed-ridden. The duration of the disease varies. Mild cases may end in recovery within a month; but the average case, as I have seen it, usually lasts for several months; while severe cases have an indefinite duration and may even leave permanent loss of power, with contractures.

Diphtheritic Polyneuritis.—The paralysis that follows diphtheria presents a special clinical type, even more marked than most forms due to other causes. Its appearance may be delayed for some days or even weeks after the membrane has cleared away from the throat, and the patient become apparently convalescent. More commonly, however, its first symptoms appear before the patient leaves his bed. The initial symptom is usually paralysis of the muscles of the palate and pharynx. This leads to nasal speech and the regurgitation of food through the nose on attempts at swallowing. An occasional early symptom also is an unusual snoring. Other early symptoms are referable to the eye. There may be paralysis of one or other of the orbital muscles: most commonly the external rectus is involved, usually in both eyes; this causes internal strabismus and diplopia, of which the patient may himself complain before the symptom is noted by others. More rarely some of the muscles supplied by the third nerve are implicated. In this case there may be ptosis and external strabismus. Power of accommodation is lost early in diphtheritic paralysis. This is perhaps one of the most constant symptoms and may occur even in cases in which other paralytic symptoms are not observed. It disables the eye for reading and for near vision, and is more annoying to, and hence more likely to be noted by adults and older children than by the very young. Another characteristic symptom in diphtheritic paralysis, especially in children, is head-drop. This is caused by paralysis of the muscles at the back of the neck, especially the trapezius. The head falls forward with the chin resting upon the sternum. I once saw this so well marked in a little boy, six years of age, that the child was unable to elevate the head without passive support beneath the chin. One of the most fatal complications of any form of multiple neuritis is the so-called heart failure of diphtheritic paralysis. This may occur suddenly in children whose cases are apparently progressing well, and who have no other symptoms of neuritis. It is the cause of

many sudden deaths in this disease. It may be preceded by a suspicious rapidity of the pulse and a great pallor of the lips and face for some hours or a day or two. In other cases the heart rate is ominously slow, the pulse falling as low as 40 or 50. This paralysis of the heart is no doubt due to interference with the functions of the pneumogastric nerve. The nerve is evidently overpowered with the toxin produced by the diphtheritic bacillus, but whether it is in a condition of acute inflammation or simply one of destruction or degeneration of its axis cylinders may be yet a question.

The extremities are usually paralyzed in diphtheritic polyneuritis, although the paralysis in the arms and legs may not be so marked as in the structures just enumerated. I have seen a child, for instance, with internal strabismus, nasal speech, head-drop, and tachycardia who was able to walk, although with a feeble gait. The paralysis does not seem to locate itself so exclusively in the extensor groups of muscles as in alcoholic cases. There is the same type of flaccid atrophic paralysis that is seen in all cases of polyneuritis. The electrical reactions are altered, although in mild cases there may not be complete reactions of degeneration. One group of muscles, however, may differ from another in this respect, owing to the fact that the poison locates itself with varying degrees of intensity in different nerves. There may be only a mild loss of faradic contractility in one set of muscles, while in another the response to faradism may be entirely abolished. Abolition of the knee-jerks is very common; in fact, the knee-jerks may be diminished or even abolished after diphtheria without marked loss of power in the legs.

The diphtheritic poison sometimes causes the ataxic or sensory type of polyneuritis. This is the type of diphtheritic pseudotabes. In this form the gait is ataxic. The sensibility of the skin of the legs and feet is diminished and there may be, although not commonly, some pain in the limbs. This form of diphtheritic paralysis, especially when it occurs in adults, may simulate locomotor ataxia. I once observed the case of a man, aged sixty years, who, after severe sore throat which had evidently been diphtheritic, had anæsthesia with numbness and tingling in the feet and legs and a very ataxic gait. In his case a diagnosis of locomotor ataxia had been made by a surgeon in attendance. Exact tests revealed slight muscular wasting and paresis, especially in the muscles below the knees. The knee jerks were entirely abolished. As an important point for differentiation in these cases, it is worth while to recall the fact that severe pain in the affected nerves and muscles is not common in diphtheritic paralysis. Certainly nothing like the fulgurant pains of tabes occurs in this disease. The bladder and rectum are not affected, as

rule. The sexual power may be diminished or even lost in severe cases. Paralysis of the phrenic nerve and of the muscles of respiration has been noted in diphtheritic paralysis.

It should be borne in mind that the diphtheritic poison is not limited in its effects entirely to the peripheral nervous system. Small hemorrhages and foci of softening have been observed in the brain and in the nuclei of the cranial nerves. Thus hemiplegia has been noted as the result of a lesion in the internal capsule. Mendell has reported such a case. Nuclear lesions occur and cause paralysis of some cranial nerves, which may be permanent; and Déjerine and others have observed alterations in the anterior horn of the gray matter of the spinal cord.

The course of diphtheritic paralysis is usually rapid and the prognosis in the majority of cases is good. The possibility of sudden death, however, from paralysis of the heart should be borne in mind.

Polyneuritis due to Lead.—The common form of lead palsy, as is well known, is that in which the extensor muscles of the wrist and hand and fingers are involved. This comes properly within the definition of a peripheral neuritis, but not so justly within that of a polyneuritis, as practically but one nerve trunk or even only one branch of that trunk is involved. As, however, the paralysis is symmetrical or bilateral, and as, moreover, lead does undoubtedly cause inflammation of other nerves in exceptional cases, this particular paralysis may be described here.

The wrist-drop or hand-drop of lead palsy is caused by a paralysis of the extensor communis digitorum, the extensor primi internodii pollicis and extensor secundi internodii pollicis, the extensor minimi digiti and the extensor indicis, the extensor carpi ulnaris, and the extensor carpi radialis longior and brevior. A peculiarity of lead palsy is that in some cases it permits the patient to extend the hand upon the wrist when the fingers are flexed. The flexor muscles of the hand are not paralyzed, although their functions are improperly performed. This is due to the physiological law already explained on a preceding page, according to which, when any muscular group performs a coördinate movement, its opposing group joins in that movement. Thus in the normal action of the flexors of the hand, it is necessary that the extensors should contract slightly and, as it were, steady the arm and hand as though for a basis of action. When this power of the extensors is lost, as in lead palsy, the flexor muscles act in an awkward, feeble, and incoördinate manner.

In lead palsy the extensor muscle of the metacarpal bone of the thumb and the supinator longus muscle are not paralyzed. These important exceptions are characteristic of this form only of polyneu-

ritis; hence they have great importance for differentiation. Sensation, as a rule, is not affected in this limited form of lead palsy. The type of paralysis, as in other forms of polyneuritis, is the flaccid and atrophic. Atrophy of the muscles occurs promptly and proceeds to an extreme degree. The back of the forearms consequently becomes flattened and apparently devoid of muscular tissue. The reactions of degeneration show themselves usually in all their classical phases, although in mild cases there may be merely a partial reaction of degeneration. The muscles of the forearm that are affected in lead poisoning are supplied, with one exception, by the posterior interosseous nerve—not by the radial, as is commonly said. The exception is the extensor carpi radialis longior, which is supplied by a branch from the musculospiral. The radial is a sensory nerve.

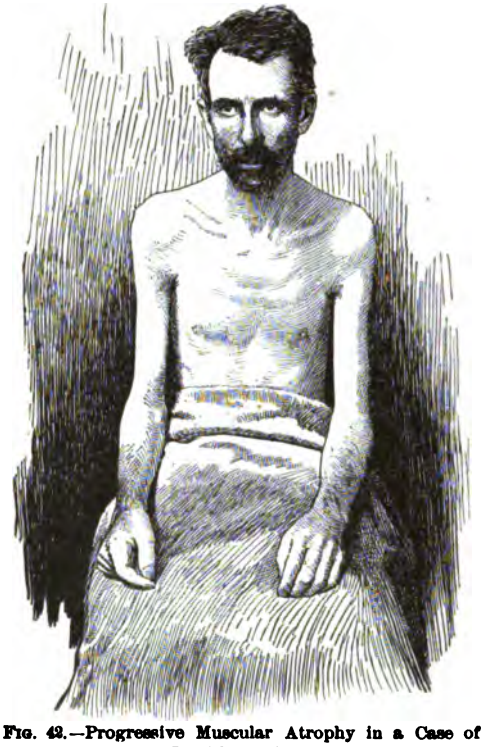

Fig. 42.—Progressive Muscular Atrophy in a Case of Lead Poisoning.

More widespread paralysis is sometimes seen in cases of lead palsy. Muscles commonly affected are the deltoid, biceps, brachialis anticus, and supinator longus. This is the upper-arm type of paralysis of Duchenne. These muscles have some physiological and anatomical, as well as pathological, association, for as Duchenne showed they can be all made to react in unison to an electrode placed at a certain point just above the clavicle. Paralysis of the nerves and muscles of the leg also may occur in lead poisoning. Some of the thigh muscles may be affected, but the muscles most likely to suffer are the extensor muscles proper below the knee—*i.e.*, the extensors of the toes and the peroneal group. The tibialis anticus is said to escape just as does the supinator longus in the arm.

The polyneuritis due to lead is especially characterized by the absence of pain, in this respect presenting a marked contrast to alco

holic neuritis. While arthralgic pains are not uncommon in chronic lead poisoning, they must not be mistaken for the pains of neuritis. Even at the onset of paralytic symptoms, pain in the nerve trunks and in the paralyzed muscles is not common. Anæsthesia is also, as a rule, conspicuous by its absence, although a slight numbness, or even loss or retardation of sensation, may be noted occasionally in the area of distribution of the radial nerve. Cramps are sometimes observed in the legs at the onset of lead palsy. They are not com-

FIG. 43.—Same as Fig. 42.

monly seen, however, in the arms. Tremor is a rare symptom in lead poisoning, and especially in cases in which paralysis has occurred. While the two symptoms are not incompatible with each other, they do not commonly coexist, in my observation, in any form of multiple neuritis. The ataxic type of multiple neuritis, the so-called pseudo-tabes, is occasionally seen in lead poisoning. I have recorded such a case, to which reference has already been made.[17] In these cases a diminution or alteration of the sensory function may sometimes be detected by careful examination. In the case referred to,

anæsthesia was observed in the legs, the hands, and the soles of the feet. The legs and arms were slightly contractured. The external and internal eye muscles were normal. Sexual power was normal and the control of the bladder was perfect. The gait was ataxic, but flapping of the feet was not marked. The hands and feet were slightly œdematous. There was partial reaction of degeneration and abolition of faradic contractility in all the paretic muscles. Optic atrophy was noted by Dr. Gould, no doubt the result of a preceding optic neuritis. The diagnosis was of chronic lead poisoning, with multiple neuritis and contracted kidney. At the autopsy no lesion was found in the spinal cord, but microscopical examination revealed the presence of neuritis. The absence of the flapping gait in this patient, so characteristic of locomotor ataxia, was due to a slight foot-drop, which obliged the patient to lift his feet high from the ground and bring them down with the toes first.

A type of progressive muscular atrophy is sometimes noted in lead poisoning. Slow progressive wasting with fibrillation is seen, and this accompanies instead of succeeds the paralysis. I have recorded briefly such a case in my article on "Diseases of Occupations," page 364, Vol. III., of this series. The man was a workman in a white-lead factory. He had extensive muscular atrophy, involving the muscles of the arms and shoulders, and also, to a less extent, those of the legs. The arms were wellnigh powerless, but the muscles had not lost their electrical irritability. Sensation was not involved. Since that report this patient has largely recovered the use of his arms. (See Figs. 42 and 43.)

Other rare types of paralysis may occur in lead palsy. Remak[391] reports an instance of mononeuritis multiplex or progressive multiple localized neuritis. It occurred in a typesetter, and began with amyotrophic paresis and reactions of degeneration in the muscles innervated by the left ulnar nerve. There was no history of lead poisoning, although the patient's occupation strongly suggests the possibility of this. Three months later paralysis with paræsthesia began in the right thigh. This was caused by localized peripheral neuritis in the motor nerves to the iliopsoas muscle and in the anterior crural and obturator nerves. The process was shown to be peripheral by the involvement of the sensory fibres of the anterior crural nerve and by paralysis of the sartorius muscle, which does not occur in poliomyelitis, and by the normal state of the tibialis anticus. The ulnar nerve of the right hand was next affected. Remak thinks that the course of the disease justifies the diagnosis of polyneuritis, but this neuritis seems to have been distributed in only well-defined areas and was not general. Remak argues, not very conclusively, that lead was not

the cause of the neuritis, because this progressed after the man had stopped working with type, and because lead palsy, in the rare cases in which it appears in the lower extremities, never attacks the crural muscles alone. The peculiarity of the case consists in its somewhat erratic course and want of symmetry, and the name mononeuritis multiplex has been suggested for it.

Fraenkel[222] reports a case of multiple neuritis in which the left facial nerve was involved and in which tumors of the skin were present in the extremities. These developed in the early stages of the disease and seemed, therefore, to have some relationship with it. They were arranged symmetrically. Histologically they consisted of small round cells, larger epithelioid cells, and spindle-shaped and giant cells. The tubercle bacillus could not be demonstrated in these tumors and inoculation gave negative results. There was no history nor sign of syphilis. Fraenkel claims that gummata of the skin, when not successfully treated, will usually soften and ulcerate or occasionally contract, in consequence of caseation at the centre. In this patient, contraction of the tumors was observed, apparently as a result of treatment. It has not been positively proven, however, according to Fraenkel, that syphilis causes multiple neuritis. This patient had been exposed to lead poisoning, but Fraenkel argues that the facial paralysis, the severe and spontaneous pain, the sensitiveness to pressure of the nerves, skin, and muscles, are difficult to explain as symptoms of lead intoxication. Such granulomata of the skin are certainly unknown in lead poisoning and facial paralysis is rare in these cases. The possibility of a rheumatic origin of the tumors is suggested. Whatever their origin, they seemed to have been due to the same poison that caused the neuritis. Fraenkel, however, is inclined to attribute the growths to syphilis.

According to Babinski paralysis of the diaphragm, of the intercostal muscles, and of the laryngeal muscles may occur in some cases of acute neuritis due to lead. These are alarming complications and may end in death, but they are rare.

The Polyneuritis of Leprosy.—One type of leprosy, as already explained, is the anæsthetic form, or the trophoneurotic leprosy of Leloir. This is marked by a prodromal period in which there may be, according to Leloir, fever, a tendency to sleep, painful gastric symptoms, and rheumatoid and neuralgic pains. Anæsthesia is gradually established. Later there is an eruptive stage in which extensive maculæ form. Trophic lesions occur, such as extensive destruction of the fingers and toes. With the anæsthesia there usually occurs some degree of paralysis and muscular atrophy, with which there may appear either partial or complete reactions of de-

generation. The resemblance of some forms of anæsthetic leprosy to the form of syringomyelia originally described by Morvan, and called by his name, has been already referred to (see page 95). Leprous neuritis is characterized by a very chronic and progressive course. It depends upon an adventitious inflammation in which the sheaths of the nerve and of the fasciculi are greatly thickened. This eventually causes pressure upon and destruction of the axis cylinders.

Beriberi.—This disease has for its chief characteristic, as already explained on a preceding page, an inflammation of the nerves. Beriberi is of infectious origin and occurs especially in China, Japan, and India, although in recent years it has been noted not infrequently in other regions of the globe. It is generally supposed to be due to a microbe, although improper and insufficient food is evidently an active factor in its causation. Thus in the case of three men observed in the Philadelphia Hospital, reported by Dercum,[221a] a diet of old salt beef, most of which was spoilt, poor bread, and occasionally beans, while the patients were working in the phosphate beds in one of the West India islands, had evidently been a potent cause. The disease is frequently associated with intestinal parasites, although these have not been proved to have an etiological relationship.

There are several types or forms of beriberi. In the very malignant form febrile symptoms with anasarca predominate. In the neurotic form the symptoms of peripheral neuritis are well marked. These are numbness, anæsthesia, paresis, loss of the deep reflexes, muscular atrophy, and more or less general anasarca. The œdema of the legs is a notable symptom. Balz and Scheube originally demonstrated that the essential lesion in this disease consists in a multiple peripheral neuritis. Other observers, notably Winkler, Musso, and Morelli, have claimed that they have found a microorganism which is the cause of the disease. Some writers have advanced the opinion, with apparently good reason, that beriberi is not an infectious disease, but is due to the use of bad food, especially poison coming from the use of certain kinds of fish. It has been found that careful regulation of the diet and the substitution of eggs, fresh meat, and vegetables for a diet of fish and rice have reduced the number of cases in the Japanese navy to a remarkable extent.

Diabetic Neuritis.—Neuralgic pains of a severe type have long been noted in diabetes. It is now well known that these are caused by irritation or inflammation of the nerves. The symptoms of involvement of the nervous system in diabetes are numerous. The cerebral intoxication, causing coma, is the most conspicuous and dreaded of these. It is probably caused by the presence in the blood

of acetone or of some other substance generated in the retrograde metamorphosis of the sugar compounds. This same substance is probably the cause of the inflammation of the nerves which is occasionally seen. The affection is rather more common in the legs than in the arms. Pain of a sharp, lancinating character, or intense and burning, is complained of. Anæsthesia may be present, and this may cause the ataxic type or the pseudotabes seen in this as in other forms of neuritis. Paralysis and atrophy of the muscles are sometimes seen. The muscles most affected are the extensor muscles of the feet and toes. The palsy of these muscles causes the high stepping gait with the drooping foot, which distinguishes pseudotabes from true locomotor ataxia. The knee jerks are lost. The following illustrative case occurred in my service at the Methodist Hospital:

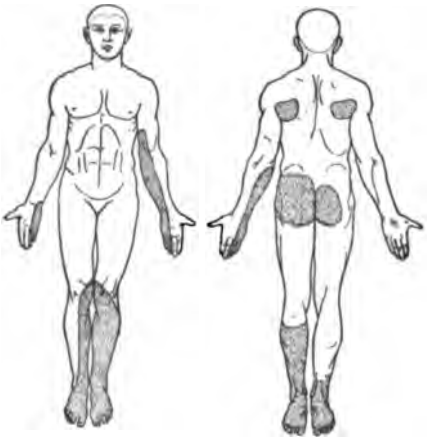

FIG. 44.—Anæsthesia in Diabetic Neuritis. (Methodist Hospital.)

E. R——, white, woman, aged 61, had suffered with diabetes for a number of years. Just before admission to the hospital she had an attack of pleurisy which had reduced her greatly. She was a thin, poorly nourished woman, who complained of nothing but general debility. She had great thirst and a ravenous appetite, in spite of which she had lost over sixty pounds. The urine contained about five per cent. of sugar and had a specific gravity of 1.040. The quantity of urine voided in the twenty-four hours varied from one hundred to one hundred and fifty-seven ounces. This patient's nervous symptoms began with pain and tingling in her legs. She had pain also in the groin. There were abolition of the knee-jerks and slight tenderness on pressure over the calf muscles and the peroneal nerve. There was no distinct anæsthesia at that time, but there was slight retardation of tactile sensation. She had a burning pain along the inner aspects of the arms. Later she had a staggering gait. The thermal sense was abolished in the toes and the soles of the feet. Power in the left leg was less than in the right. The left arm was weaker than the right and the deep reflexes were wanting in both arms. Later the anæsthesia on the soles of the feet and on the toes was marked and was accompanied with incoördination. The patient swayed violently when her eyes were closed. The muscular sense was diminished in the left leg and the sensation to touch, pain, and temperature was diminished in the left leg below the knee. Some œdema of the ankle set in. The pain in the feet was so

great at times that the patient sat up nearly all night. Acetone was found in the urine. The accompanying diagram (Fig. 44) shows the areas of disturbed sensation in this patient.

Tabetic Neuritis.—As has been known for some years, locomotor ataxia, especially in its advanced stages, may be associated with a peripheral neuritis. The atrophy of the optic nerve, and even occa-

Fig. 45.—Multiple Neuritis in Locomotor Ataxia. (Philadelphia Hospital.)

sionally the involvement of the oculomotor nerve, have long been known. Bourdon[222] in 1861 was one of the first to note and describe atrophy of the oculomotor and abducens nerves in tabes. Marotte observed atrophy in the muscular branches of the gastrocnemius in a case of tabes. Subsequent observations were made by Friedreich, who found the hypoglossal, the sciatic, the crural, and the brachial nerves atrophied. Westphal also noted slight atrophy in the sciatic and tibial nerves. These results, according to Pitres and Vaillard,[223] were not accepted readily by neuropathologists, who claimed that the

atrophies noted in the nerve trunks must have been due to primary alteration in the ganglionic centres of the nerves. Pierret, however, in 1880, Déjerine in 1882, and Pitres in the same year, published results of their observations upon degeneration of peripheral nerves in tabes. It is now generally accepted that a peripheral neuritis may occur in this disease. Pitres and Vaillard, in their classical

FIG. 46.—Muscular Atrophy, Probably due to Neuritis, with Double Ptosis, in Locomotor Ataxia. (Burr.)

paper, conclude that the peripheral nerves are frequently the seat of alterations in tabes. These alterations do not differ from those observed in non-traumatic neuritis. The distribution varies widely. The changes usually begin in the terminal branches of the nerves. Their extent and gravity have no constant relation with the extent and stage of the posterior sclerosis. These authors claim, rather dogmatically, that these changes in the nerves do not figure in the

causation of the fulgurant pains in ataxia and the abolition of the knee jerks; but, on the other hand, that they may cause areas of cutaneous anæsthesia and analgesia, trophic lesions, such as perforating ulcer, and various eruptions, with dystrophy and falling of the nails; also motor paralysis, with or without muscular atrophy, and arthropathies and spontaneous fractures. These authors conclude finally that some of the visceral crises may be caused by inflammation of the visceral nerve.

The illustration on page 438 (Fig. 45) is of a tabetic patient from my clinic in the Philadelphia Hospital. She is in an advanced stage of locomotor ataxia with all the classical sensorimotor symptoms of the disease, such as incoördination, fulgurant pains, gastric crises of an aggravated type, abolished knee jerks, and optic atrophy. She has also had frequent luxation of the right shoulder-joint. She now has an advanced stage of paralysis and muscular atrophy in all the extremities and even in the trunk. There is profound anæsthesia in some of these parts. There are no arthropathies or trophic lesions of the skin, but the luxation of the shoulder is probably due to some wasting and weakness of the ligaments of the joint. Another patient (Fig. 46) from Burr's clinic in the same hospital has marked muscular atrophy, probably due to neuritis, and also a characteristic double tabetic ptosis.

Arsenical Polyneuritis.—This form is characterized especially by pain, anæsthesia, paralysis, muscular atrophy, abolished knee jerks, and ataxia. It may result from acute or chronic forms of poisoning. It presents nothing especially distinctive.

Tuberculous Neuritis.—As already explained, when describing the various causes of multiple neuritis, this affection, in a variety of grades and without constant distribution, may appear in patients suffering with tuberculosis. The nerves of the legs, especially the great sciatic, seem to be especially predisposed to the action of the poison.

Neuritis of Typhoid Fever.—In this form, as already stated, certain nerves, as the peroneal and ulnar, are especially liable to be involved. In severe cases, however, the inflammation is more widespread, as in the case already referred to, of which an illustration is presented on the following page.

The Polyneuritis of Influenza.—According to Putnam[220a] multiple neuritis, often of severe onset, may follow influenza. Optic neuritis may occur early. Other nervous lesions, such as meningitis, encephalitis, and myelitis, may complicate the clinical picture. This form of polyneuritis is undoubtedly very rare.

In describing the above clinical forms, I have had reference

chiefly to their etiology. Multiple neuritis, however, presents various types which are not caused exclusively by any one particular poison. The most conspicuous of these is the *ataxic* type, which is caused by various poisons, as alcohol, lead, and the poisons of diphtheria and diabetes. It has already been specially described in the preceding pages.

Another form sometimes seen is called *neuritis migrans*. This is characterized by a slowly progressive neuritis extending or passing from one set of nerves to another. It usually has a very chronic course.

Lunz[224] relates a case in which curious atrophic symptoms were probably due to a neuritis migrans. This peculiar affection occurrred in a woman, and dated from the period of her marriage and exposure in field work. The first symptom was periodic pain in the

FIG. 47.—Multiple Neuritis following Typhoid Fever. (Methodist Hospital.)

posterior part of the right thigh, radiating into the sole of the foot. In about three years wasting of the right lower limb began, and the patient noticed that the right breast was smaller than the left and secreted less milk. Still later, the left cheek began to atrophy and at the same time pain was experienced in it. Pains were noted also in the back and in the left lower limb, as well as in the regions of the right scapula and right upper limb. When Lunz examined the patient, whose disease had then lasted about nine years, the wasting in the skin and muscles of the left cheek was marked. Atrophy was also noted at the inner border of the scapula, on the right side of the abdomen, and in the right lower extremity. There was no true paralysis, but the right leg gave out sooner than the left in walking. Pain was increased by movement. Pressure over the lumbar and sacral vertebræ and over the nerve trunks and muscles of the right lower limb caused sensations of pain. There was no anæsthesia or reaction of degeneration. The patella reflexes were exaggerated. As the hyper-

æsthesia and atrophy were limited distinctly to regions innervated by certain nerves, the disease cannot be regarded as central. Lunz believes that it was a neuritis migrans, and that the atrophy was probably the result of vasomotor disturbance. It is scarcely necessary to follow Lunz in his speculations on the vasomotor origin of some of the symptoms in the case of his patient. He reasons that the atrophy following joint disease can be explained by the irritation of sensory nerves, which acts on the vasomotor centres in such a way that vascular constriction is caused. This patient also had attacks of headache, vertigo, tinnitus aurium, sensations of heat and cold, and free perspiration, all of which Lunz believes were due to vasomotor disturbance.

Diagnosis.

Multiple neuritis requires to be distinguished especially from locomotor ataxia, some forms of myelitis, the muscular dystrophies, syringomyelia, insular sclerosis, and hysteria.

From *locomotor ataxia* it is not always easy to distinguish multiple neuritis. Mistakes are made more readily perhaps between these two diseases than between multiple neuritis and any other affection. This is true especially of the sensory or ataxic type of polyneuritis. Locomotor ataxia differs from multiple neuritis in its mode of onset. The fulgurant pains are lightning-like, spontaneous, and more intermittent than the pains of multiple neuritis. Moreover, they are not aggravated by pressure upon the nerve trunks and the muscles. Paralytic phenomena are not seen early in tabes. The muscles do not atrophy and present reactions of degeneration, except in the rare instances in which a peripheral neuritis is associated with a posterior sclerosis. Ataxia and swaying with the eyes shut are common symptoms both in tabes and in the pseudotabes of polyneuritis, but in the latter disease there is usually some degree of paresis and degeneration of the muscles. Hence the gait differs in the two affections. In tabes the gait is wide and flapping, the foot striking first with the heel. In pseudotabes the gait is high-stepping, with the foot dropping and striking first with the toes. This is due to the loss of power in the peroneal and extensor muscles. In some few instances, it is true, this gait of "steppage" is not marked in pseudotabes. In locomotor ataxia early paresis of the bladder and impairment of the sexual power are often seen. These do not occur in multiple neuritis. The visceral crises of tabes are not simulated by any symptom in multiple neuritis, unless it be the pains of gastric catarrh in alcoholic patients. These latter, however, have not the spontaneous and intermittent character of the crises, and they are increased on pressure. The

differences in the ocular symptoms are manifest. In locomotor ataxia optic atrophy is not uncommon while the characteristic Argyll-Robertson pupil is frequently seen. This consists of a paralysis of the iris to light, with preservation of its contractility on accommodation. Nothing like this is seen in any form of multiple neuritis. The claim made by Eperon, that he had found the Argyll-Robertson pupil, with other tabetiform symptoms, in patients suffering with toxic ambylopia and symptoms of multiple neuritis, has already been referred to (see page 425). From his own reports there can be little doubt that the patients had posterior sclerosis. In diphtheritic neuritis there may be paralysis of accommodation, without paralysis of the iris. In multiple neuritis, it is true, there may be optic neuritis or post-neuritic atrophy in patients who have long been exposed to lead, alcohol, and tobacco; but this symptom is not associated with the Argyll-Robertson pupil. The history in such cases also throws light upon the diagnosis. Finally, multiple neuritis, as a rule, is a disease of much more rapid onset than locomotor ataxia. In severe cases the patient may pass from a condition of comparative health to one in which he is confined to bed totally paralyzed. Locomotor ataxia, it is true, may sometimes have a rapid evolution, and the early stage in these cases may closely simulate multiple neuritis. Thus I have reported a case of acute locomotor ataxia[52] in a ship's carpenter, which began apparently as an acute multiple neuritis caused by the patient becoming overheated on shipboard, while crossing the equator, and then having himself drenched with buckets of cool water. The symptoms which supervened in a few days were motor paralysis and anæsthesia of the extremities; but later the patient developed well-marked tabetic symptoms, with arthropathy of the ankle-joint. Such a case raises the question whether acute multiple neuritis may be the starting-point for an inflammation of the posterior column of the spinal cord. I have made several observations which tend to support this view. Riesmann and the writer[105] have reported a case of endocarditis with septicæmia and multiple neuritis, already referred to in this article, in which there was degeneration of the posterior column of the spinal cord. Trophic lesions, such as perforating ulcer and arthropathies, are seen in tabes, but not in multiple neuritis. In the latter disease muscular atrophy and œdema are the chief trophic symptoms. Finally, the etiology of the two diseases differs. Tabetic patients very frequently have a history of syphilis; patients with multiple neuritis have usually a history of exposure to some poison or toxin, possibly associated, especially in alcoholic cases, with privation and exposure to cold and wet.

(see Fig. 45), the neuritis has been slowly progressive for several years and still the course of the patient's primary disease does not seem to be materially hastened by it. She is, of course, entirely confined to bed, and this fact has seemed to me to be rather conservative than otherwise in her case.

Another class of causes comprises alcohol, diphtheria, lead, and the other metallic poisons. These poisons do not necessarily excite a fatal neuritis. In estimating the prognosis in any of these cases, the cause itself sinks into comparative insignificance compared with the severity of the symptoms and concomitant circumstances. In alcoholic cases it is not always the neuritis itself that kills. Pulmonary, cardiac, hepatic, and renal complications may have a determining effect. The most serious complications depending on the neuritis itself are enfeeblement of the heart and embarrassment of respiration. In cases in which advanced tachycardia is present and the expansion of the chest during inspiration is much impaired, a guarded prognosis should be given. These symptoms, however, affect the prognosis as to life rather than duration. In other words, the patient may escape with his life in cases in which these symptoms have been threatening and yet he may have a most protracted course of paralysis with very slow recovery. The duration, in fact, of these alcoholic cases is sometimes most protracted. It is not unusual for the patients in severe cases to remain paralyzed, and even bedridden, for many months. I have known several cases in which little progress had been made even after a year's treatment in the hospital. In some instances, in fact, the patient with alcoholic neuritis may be permanently paralyzed, with contractures at the knee-joints. In alcoholic cases, again, the mode of onset has not a little to do with the prognosis. Thus cases with a sudden or acute onset are much more likely to present dangerous complications than cases in which the onset is slower and more insidious. This of course is simply saying, in other words, that in severe and pernicious cases the poison acts with more rapidity and malignancy than in milder cases.

In postdiphtheritic paralysis the prognosis depends upon a variety of circumstances. In cases which occur early—*i.e.*, before the throat is cleared and convalescence has well begun—the issue is more doubtful than in cases the onset of which appears several weeks later. The chief complication to be feared, however, is paralysis of the heart. Extreme rapidity of the heart, with impairment of the first sound and weakness of the impulse against the chest wall, and with pallor of the skin and mucous membrane, is always a sign of grave import. It does not necessarily betoken, however, a fatal issue. The opposite condition or bradycardia, in

which the heart beat is much diminished in frequency, sometimes to as low as 50 or even 40 to the minute, should also lead to a guarded prognosis. It is by heart failure, in fact, that diphtheritic paralysis usually kills. If the patient escapes this danger the recovery is almost without a question assured. Exceptions to this rule may occur in cases in which there is very widespread paralysis and in which palsy of the palate, pharynx, and possibly even the tongue renders swallowing difficult or even impossible. In such cases general prostration rapidly supervenes and the patients die of exhaustion.

Lead poisoning seldom destroys life by involvement of the peripheral nervous system. Very acute, pernicious cases of widespread paralysis have, however, been reported, in which death has occurred. Such cases are extremely rare. The danger in lead palsy is of a more or less protracted muscular atrophy with corresponding loss of power, especially in the extensor muscles of the forearm. This paralysis may last for years—and I have seen cases which gave every promise of being permanent. In the more widespread muscular atrophy of the type already described and illustrated (see page 434), the course is very chronic and the duration uncertain. In the case referred to, however, I have seen progressive improvement, and some of the paralyzed and wasted muscles have been restored, although the patient is still in the hospital after a period of three years.

In arsenical paralysis recovery is not rapid. In acute cases in which the poison has been taken by the stomach, the danger, however, is from the gastrointestinal irritation. Secondly, the severity of the case, from whatever cause it arises, has, as we have already said, much to do with the prognosis. In cases in which the onset is acute and the disease widespread, the dangers to life are much greater than in cases in which the disease has a more insidious onset and a more chronic course.

Finally, the conditions and circumstances of the patient have something to do with the prognosis. In women who have been much reduced by hard work, exposure, poor food, and over-childbearing, the prospects are that the course of the disease, even if the patient escapes with her life, will be much protracted. I am not sure, however, that sex itself materially influences the prognosis. Men, who have been much exposed and reduced, offer as little resistance to the disease as do women. Hence patients among the poorer, and particularly the pauper classes, are especially likely to suffer from a protracted course of the disease. After any one of the infectious diseases, by which the patient's strength has been much reduced, convalescence is much delayed by an attack of neuritis, and

the paralysis itself usually pursues a chronic course. This depends, of course, upon the extent of the distribution of the neuritis. In cases in which only one or a few nerves are involved, as for instance the peroneal and ulnar nerves in typhoid fever, recovery may be satisfactory after a comparatively short course.

Morbid Anatomy.

I have already discussed, in a preceding section of this article (see page 42), the general subject of the pathological anatomy of nerves. In that section the anatomy of neuritis was described, and reference can be made to it for details, which need not be repeated here.

Morbid anatomists usually describe two varieties of neuritis; first, the *parenchymatous*, and, second, the *interstitial*. As Mme. Dejerine-Klumpke[111] has well said, whatever may be the cause of polyneuritis, its pathological anatomy is nearly always the same. According to this observer, the lesions reproduce in effect in the great majority of cases the well-known characteristics of parenchymatous neuritis, *i.e.*, segmentation of the myelin, proliferation of the nuclei in the sheath of Schwann, destruction of the axis cylinder, and final atrophy, more or less complete, of the nerve fibres. This, in brief, is the clinical picture presented by the ideal parenchymatous neuritis. It does not, however, take into account sufficiently, in my opinion, the alterations in the blood-vessels and in the connective tissue which characterize even this type of cases. The fault in the classical distinctions, too much insisted on by some pathologists, between the parenchymatous and interstitial varieties of neuritis is that in describing the former they too much ignore the vascular and interstitial elements of the nerves. As we have seen, in discussing the etiology of polyneuritis, this disease is usually caused by some irritant or poison circulating in the blood. It must, therefore, reach the nerve fibre through the small blood-vessels, and it is not unreasonable to suppose that these will react against the poison just as the neuron does. Hence some thickening of the minute blood-vessels and proliferation of connective tissue is to be looked for even in cases of parenchymatous neuritis.

The pure type of interstitial neuritis is marked by proliferation of the connective tissue of the nerve, in some cases by thickening of its sheath, by congestion and swelling of the nerve trunk, dilatation of its vessels, extravasations of blood, and even, in some rare cases, by formation of pus. But in these cases the lesions certainly do not respect the nerve tubules and their contents. These are pressed upon and in many instances destroyed. Usually the myelin is

broken up, the axis cylinder is destroyed, and the nuclei in the sheath of Schwann proliferate. This type of neuritis has been claimed by some observers to be seen almost exclusively in the acute and rapidly fatal forms of neuritis. This claim is criticised by Dejerine-Klumpke, who very justly points out the fact that parenchymatous lesions, while less pronounced, are not absent. These are segmentation and disappearance of the myelin, with destruction of the axis cylinders. From my own observations, it seems to me to be an exaggerated claim that the ordinary cases of multiple neuritis preserve a purely parenchymatous type. It may be true, however, that in ordinary polyneuritis the nerve tubule and its contents, the myelin and axis cylinder, suffer first and most from the poison diffused into them from the blood; but certainly, in advanced cases at least, the evidences of involvement of the connective tissue are not lacking.

In pure parenchymatous neuritis, changes are not visible to the naked eye. Under the microscope, however, the disappearance of the axis cylinders and the segmentation of the myelin are very marked. In the interstitial type of neuritis the nerve may be perceptibly thickened and altered in texture, even to naked-eye inspection. Under the microscope the connective tissue is seen to be enormously increased, lying in concentric lamina about the fasciculi. The blood-vessels are also thickened and usually crowded with corpuscles. Free leucocytes may be found in the tissues of the nerve, although the formation of pus is not common. This was distinctly the type of neuritis observed by me in the case of septicæmia already referred to. In that case the nerve fibres were also involved in a degenerative or destructive process. Many of the axis cylinders had disappeared.

The formation of pus is more likely to occur in cases of isolated neuritis, for instance, as after trauma. The type of inflammation in these cases is distinctly hemorrhagic and interstitial. In one case of sciatica I saw this type of inflammation well marked. The nerve was perceptibly swollen and congested; blood and pus were extravasated through its structures.

It is possible that some pathologists, who have so strongly insisted upon a purely parenchymatous neuritis, have been merely looking upon a Wallerian degeneration of the axis cylinder of the neuron, which had resulted from a more distinctly inflammatory lesion in some other part of its course. Certainly, from their descriptions, the parenchymatous neuritis exactly simulated a degeneration of the axis cylinder. Babinski calls attention to this fact in discussing the absence of distinct or special lesions due to the various causes of neuritis.

Gombault [227] has described what he claims to be a special type of neuritis due to lead. This is the so-called *periaxial* neuritis. It is characterized by the preservation of the axis cylinder, with a grouping of the fragments of myelin along its course. This preservation of the axis cylinder seems to be a special characteristic of this type of cases. In some cases the nerve fibre is slender and of small calibre. These lesions in cases of lead poisoning, according to Gombault, are irregularly disseminated. According to Dejerine-Klumpke, the periaxial form of neuritis predominates in cases caused by mercury.

In leprous neuritis, the adventitial or interstitial type is common. The sheaths of the nerves and the intrafascicular connective tissue are often enormously thickened.

According to Abraham, [228a] the median, ulnar, posterior tibial, and peroneal nerves are most commonly affected. The facial and radial rank next in liability to attack. In some cases the recurrent laryngeal nerve is attacked, and this causes aphonia. The affected nerves are the seats of fusiform swellings, which are reddish-gray in color and translucent and gelatinous in appearance. These may be from two to four times the diameter of the normal nerve. They are often larger at places where the nerve is superficial, as behind the internal condyle of the humerus or below the head of the fibula. These swellings are due to a perineuritis, in which there is a proliferation of the so-called "lepra" cells. The axis cylinders degenerate, but this is apparently a secondary process.

In alcoholic multiple neuritis, the consensus of opinion seems to be that the parenchymatous type predominates, although most authors describe a mixed type in which the connective tissue, as well as the axis cylinders, are involved. This is in accordance, also, with my own observations. It seems probable that alcohol acts primarily on the axis cylinder of the neuron; as this degenerates and dies, a secondary inflammatory process takes place in the connnective tissue. It is an interesting question, in this connection, whether the products of retrograde metamorphosis in the degenerating axis cylinder and the segmenting myelin may not constitute irritant substances which act still further to promote an inflammatory reaction in the connective-tissue elements.

In diphtheritic paralysis, the change is usually limited largely to the nerve fibres; hence it preserves a type of parenchymatous neuritis. The toxin of the disease evidently acts primarily upon the neuron and its axis cylinder, and the evidence seems to be that the latter is not alone affected, for the degenerative or inflammatory process is observed to extend up to the roots of the nerves. Moreover, in many cases, as is well known, the nuclei of the nerves (*i.e.*, the cell

bodies of the peripheral neurons) are involved. According to most observers, the interstitial connective tissue is not involved to a marked extent in postdiphtheritic neuritis.

The distribution of the neuritis varies in different cases. In the alcoholic cases, the terminal branches and the peripheral portions of the nerves are most involved. The inflammation extends up along the nerve trunk to a variable extent, but it usually ceases entirely some distance below the spinal roots. In diphtheritic paralysis, however, as already said, the inflammatory or destructive process may involve the whole neuron. In some forms of neuritis, especially that due to lead, the distribution is segmental in type, *i.e.*, various portions or segments only are involved and these may not be necessarily contiguous segments.

The changes in the muscles are usually those of degeneration with loss of striation of the muscular fibres and some proliferation of the connective-tissue element.

The spinal cord is usually not involved in typical cases of multiple neuritis. It is yet a question, however, whether the acute infectious poisons do not involve the whole of the neuron, in certain cases, and hence invade to some extent the spinal cord. Berkely's investigations upon the action of ricin and other poisons seem to show that the whole neuron is involved; and this may yet be proved to be true in some cases of multiple neuritis. In a case of septicæmia already referred to I once observed degeneration of the posterior columns of the spinal cord in association with a widespread multiple neuritis.

Treatment.

The first and most important indication in the treatment of multiple neuritis is to remove the cause. This subject, in some cases especially, is not to be slighted or ignored. Alcoholic patients require careful supervision in this respect. This is true of women in private practice, rather than of hospital cases, for women in their own households, if not carefully guarded by reliable attendants, will often find a way to obtain surreptitiously the coveted drink. Thus the best plan of treatment may be frustrated. I have known a woman to bribe a negro servant to bring her whiskey when she was bedridden with paralysis. In the case of the metallic poisons, the utmost care should be taken to avoid a continued action of the cause; or, what is more likely to occur, a return to the circumstances which entail an exposure to the metal. Thus, in cases of lead palsy in workmen, it is often difficult to prevent the patients from returning to their work as soon as their worst symptoms have disappeared. Hence a

relapse is likely to occur, and the second attack will probably prove worse than the first. I have frequently seen such cases in hospital practice, so that I have become impressed with the necessity of advising workmen who are afflicted with neuritis due to lead to abandon the work altogether. It is only by this course that many of them can escape permanent disability.

Other important indications in treatment are to relieve pain, to promote elimination, to control the inflammatory action in the nerve trunks, to guard the heart and respiration, to prevent contractures, to promote repair, and to preserve the patient's strength and nutrition.

Too much stress should not be laid upon the necessity of relieving pain. A certain amount of pain is inevitable in these cases, and if the attempt is made to keep this symptom under thorough control, it will result in the patient being more or less constantly under the influence of sedative drugs. Hence the danger will arise of his simply substituting one bad habit for another. Alcoholic patients especially will soon become entirely dependent upon these drugs. As the pains in the active stage of the disease may continue for some weeks, they cannot be controlled with drugs without a positive risk of permanently injuring such patients. Therefore I advise great caution in the use of morphine or any opiate in alcoholic multiple neuritis. I have frequently treated these invalids successfully, and with at least a comparative degree of comfort to themselves, without the use of any opiate whatever. In my observation this one drug is more fraught with mischief to such patients than all other drugs together, unless it be the equally pernicious cocaine. For the relief of pain, rest in bed and proper support for the legs and feet are in themselves sovereign remedies. If they are properly used, the patient, although perhaps not absolutely relieved, is nevertheless made fairly comfortable and kept in a much better condition for recovery than when his or her system is still further devastated by opium and cocaine. To promote this rest, the legs should be swathed in flannel bandages and the feet enveloped in cotton wool. This last precaution is especially indicated in those cases in which the soles of the feet are exquisitely sensitive. This cotton dressing promotes warmth and free sweating, and both of these are beneficial to the sore and paralyzed parts. In case anodyne drugs are deemed absolutely essential, however (it is only in alcoholic cases that they will be likely to be so), there can be no doubt that morphine is the most efficacious. If used, it should be given under the skin, and it is best if possible not to let the patient know what drug he or she is getting. Small doses only should be used (from one-eighth to one-sixth of a grain), so as not to keep the patient too deeply under the influence of the drug.

Morphine should not be used by rote, but only to meet an urgent indication; and better if possible in the evening, so as to promote a night's sleep. It should be kept strictly under the control of the physician and given only by his hand, and its use should be suspended or entirely stopped at the earliest possible moment. Cocaine is also a potent drug to relieve pain, but it has already done so much evil to the slaves of habit that its use should be entirely interdicted in alcoholic patients. The coal-tar preparations, such as antipyrin, phenacetin, and exalgin, are of less value. They tend to deplete the blood, and are not very efficient as analgesics for patients with inflamed nerves. Finally, it should always be borne in mind that time tends to relieve this symptom, and that this time will be perceptibly shortened if the patient's system is kept in the best possible condition by promoting the assimilation of food.

To promote elimination, especially in the early stages of the disease, when the patient's system may still be saturated with the poison, various drugs are of use according to the case. In alcoholic cases mild diaphoretics and diuretics are of value, especially if the action of the kidneys and liver is impaired. Hot diluent drinks, such as lemonade not too highly sweetened, and the ordinary spirits of Mindererus, are indicated. If there is albuminuria or an impaired excretion of the solids of the urine, I am fond of using a weak preparation of Basham's mixture of acetate of iron and ammonium, even in the early stages. In cases of lead poisoning the early administration of sulphuric acid and the iodides is strongly indicated. Warm baths in patients who are not too weak are beneficial. They tend to restore circulation in the extremities and promote a free action of the skin.

To control the inflammatory action in the nerve trunks, we have no real specific. The rest in bed and enveloping the extremities in flannel and cotton are probably after all as efficient for this purpose as any drugs can be. Many authorities, however, recommend the use of the salicylates of sodium or ammonium. These are supposed to control in some way the inflammatory action in the nerves. They may, however, be very disturbing to the stomach, and thus do more harm than good. I have never assured myself, in fact, that any preparation of salicylic acid was of much use in any stage of multiple neuritis. The same may be said of iodide of potassium and of the mercurial preparations. The so-called alterative action of these drugs is very little manifested in this disease. When administered after the acute stage is passed, they are probably often credited with an improvement that is merely coincident with, and not consequent upon their use.

Special attention should be paid, particularly in grave cases, to

the action of the heart and respiratory muscles. In cases in which tachycardia is marked, complete recumbency should be enjoined. The patient's head and shoulders should not be elevated even by high pillows. This precaution is especially necessary in cases of diphtheritic paralysis. In alcoholic cases the temptation may arise to give the patient small doses of whiskey or some strong wine, but I do not believe in this treatment, as it seems contrary to reason to use as a remedy the substance that has already acted as the cause of the disease. I have relied most in these cases upon the administration of digitalis, strophanthus, caffeine, and strychnine. I have usually been impressed with the fact, however, that digitalis and strophanthus do not readily exert their full physiological action upon the heart in these cases, especially in those due to alcohol. This is probably because the pneumogastric nerve is involved in the inflammatory process, and consequently offers resistance to the action of these remedies. Strychnine under the skin in full doses (from one-twentieth to one-thirtieth of a grain in adults) is by far the most reliable stimulant for these cases. Caffeine, because of its simultaneous action upon the brain, the heart, and the kidneys, is useful. Its irritating effects upon the stomach, however, must not be ignored. In diphtheritic cases an alcoholic stimulant is urgently indicated in cases in which heart failure is threatened.

The contractures, which cause so much pain and even ultimate deformity in multiple neuritis, are not easily overcome. Patients do not bear extension well, because of the extreme sensitiveness of the legs and feet. Bandaging to a splint, applied alongside of or beneath the knees, may be tried. Such a splint usually becomes very irksome, however, and in some cases seems to increase the pain. Gentle massage and an occasional persistent passive extension of the legs will do much to prevent these contractures. Later in the case, when the painful stage has passed by, this massage and passive extension should be practised still more assiduously, and they then act very satisfactorily.

To promote the tone and power in the paralyzed muscles when the stage of recovery has begun, electricity is a valuable remedy. It should not be used early in the disease, as it then increases the pain and probably tends to favor contractures. When used later, the galvanic current is likely to be the more efficacious, as the faradic contractility of the muscles may still be diminished or even abolished. As long as this contractility is impaired, the use of the faradic current is probably in vain.

Finally, every effort should be made to maintain the patient's nutrition. Special attention should be paid to the functions of the

gastrointestinal tract. If the digestion is poor, a preparation of pepsin and hydrochloric acid is indicated. The bowels should be kept active by mild laxatives and an occasional enema of warm water and soapsuds. Only the simplest, most nutritious, and most digestible food should be given, and special attention should be paid to the administration of proper quantities. These patients, especially in the early stages, often eat poorly, and their appetites must be tempted, or at least the patients should be induced to take habitually a sufficient supply.

During the stage of improvement, the use of strychnine, preferably under the skin, and of some preparation of iron is often indicated. These patients frequently are anæmic, and hence are benefited by iron. I prefer Basham's mixture, because it is easily and rapidly assimilated, and acts satisfactorily to promote elimination by the kidneys. Blaud's pills, however, are valuable, especially in women whose blood-making powers are much reduced. I do not like arsenic for these cases, because of its well-known tendency, when continued for a long time, to irritate and even inflame the nerves.

It is thus seen that the treatment of multiple neuritis is in the main simple. The disease is not one that demands inordinate dosing with a great variety of drugs. Time and patience are sovereign remedies, and will be required usually in large measure. If the patient is put in a proper state of rest and nutrition, and is protected from the injurious effects of the poison which has induced his disorder, and also from the effects of worry and excitement, it will be found usually that the natural tendency of the disease is towards recovery. The practitioner should avail himself of this tendency, and should control the natural impatience of the invalid and his friends by the exercise of proper moral influence. Even in grave cases, in which death is threatened, we have no better remedies than those indicated above.

DISEASES OF THE SYMPATHETIC NERVOUS SYSTEM. *

This system consists of a group of ganglia associated especially with the fifth nerve, and a chain of ganglia on either side of the spine, extending from the skull to the coccyx. These ganglia, particularly those along the spine, are united by intervening branches from one to the other in the series, and also by some transverse

* I have to acknowledge the valuable assistance of Dr. William G. Spiller in preparing this section on the sympathetic nervous system. The work of literary reference especially has been done by Dr. Spiller.

branches passing across from the ganglia of one side to those of the other. The ganglia are less numerous than the vertebræ, there being but three in the cervical region, twelve in the dorsal, four in the lumbar, five in the sacral, and one in the coccygeal (Gray). The chief branches of each ganglion of the sympathetic are three in number, namely, (1) branches to the neighboring ganglia of the system; (2) branches to the cerebrospinal nerves; (3) the branches of distribution, which pass either to the arteries, to the viscera, or to other large ganglia forming supplementary centres within the cavities of the body. The ultimate distribution, however, of all the terminal branches of the sympathetic system seems to be in connection with the small blood-vessels.

The chief subdivisions of the sympathetic nervous system are the cavernous plexus, cervical portion, carotid plexus, the great solar plexus, the thoracic branches with the great splanchnic nerve, and the pelvic plexus. There are numerous smaller plexuses which need not be named here. The fibres of the sympathetic nerve, as already explained, are non-medullated.

The functions of the sympathetic nervous system are varied. This system has probably largely to do with the control of the blood supply in the various parts and organs. It thus probably influences indirectly the secretion of sweat and the activity of the various glands. Among its special functions may be mentioned the following:

The cervical sympathetic controls the dilatation of the pupil; it supplies motor fibres to Müller's muscle—*i.e.*, the unstriped muscular fibres of the orbit; it controls the action of the salivary glands and of the sweat glands of the face and neck: and, finally, it sends vasomotor branches to the ear, side of the face, conjunctiva, and to the parts generally in the eye, throat, and brain and its membranes.

In the thorax, the sympathetic supplies accelerator fibres to the heart; these probably have their origin in the medulla. It gives origin to the great splanchnic nerve. In the abdomen and pelvis, in addition to its connection with the great glandular structures, it contributes motor and vasomotor fibres to the large intestine, bladder, ureters, uterus, vas deferens, and vesiculæ seminales. According to Landois and Stirling, stimulation of the abdominal sympathetic causes increased movements in these organs, while section of these nerves is followed by dilatation of the blood-vessels with derangement of the circulation and with trophic lesions. In the thorax and abdomen the sympathetic is subdivided into minor plexuses, which have special connections with the various organs. The suprarenal capsules, in which the nerves are extremely numerous, have connections with the renal and solar plexuses.

The pathology of the sympathetic nervous system is exceedingly obscure. Derangement of this system probably enters into the symptom-complex of many diseases, but the difficulty is to distinguish exactly what its particular symptoms are. Without stopping to indulge in speculation, I shall describe very briefly the few known facts about the diseases and injuries of the sympathetic.

The cervical portion of the sympathetic is the most exposed to direct injury. It may suffer from penetrating, incised, and gunshot wounds; from blows, pressure, and contusions; also from tumors, such as aneurysms, enlarged glands, exostoses, and malignant growths. According to some authors, it may be irritated by affections of the apices of the lungs and the overlying pleura. It is not usually the seat of any acute inflammatory disease, such as is caused by an infectious process or by exposure to cold. This portion of the sympathetic, however, may be involved in cerebral lesions, as has been noted by Nothnagel. Symptoms of irritation have been noted in a few cases of hemiplegia. Three very striking cases were published by Windscheid[""] of hyperidrosis, in which a pathological condition of the seventh nerve existed. Dufour[""] published observations on paralytic affections of the cervical sympathetic with incomplete closure of the palpebral cleft. I shall relate and illustrate in this paper a case of hemiplegia associated with symptoms of irritation of the cervical sympathetic on the same side. The central connections of the cervical sympathetic system, by which these symptoms are caused, are supposed to be in the optic thalamus. According to Landois and Stirling, stimulation of the cervical sympathetic in man causes dilatation of the pupil, pallor of the face, occasional hyperidrosis, disturbance of vision for near objects, and protrusion of the eyeball with widening of the palpebral fissure. Paralysis or section of this nerve, on the other hand, causes flushing of the blood-vessels on the side of the head, with occasional anidrosis, contraction of the pupil, retraction of the eyeball, and narrowing of the palpebral cleft. These symptoms are said to have occasionally accompanied unilateral atrophy of the face. In the thorax, irritation of the sympathetic strands to the heart probably causes acceleration of that organ.

My case is as follows:

J. B——, white, male, aged 45 years, a native of Switzerland, was admitted into the Philadelphia Hospital with hemiplegia. He had a history of syphilis. More than ten years before admission he had had a paralytic stroke, which was followed in a few months by a second. In each attack he lost consciousness. He never entirely recovered from the second stroke, and on admission was dull and melancholy. He was found to have a right-sided hemiplegia, with

contractures of the arm and, to a less degree, of the leg. The knee jerk was exaggerated on the right side. The patient was able to walk. Sensation was preserved. The right pupil was dilated, and the palpebral fissure was widened. His most striking symptom was the continuous sweating of the right side of the face and forehead. This sweating was limited by the median line on the forehead, face, and neck, and was closely confined to the distribution of the fifth nerve. There was not an active flushing of the face and ear, although

FIG. 48.—Unilateral Facial Hyperidrosis (right side) with Widening of the Palpebral Fissure and Dilatation of the Pupil, caused by Irritation of the Sympathetic Nerves. (Philadelphia Hospital.)

the complexion was darker on the affected side. The perspiration stood in large beads on the affected area, and apparently was not influenced by changes in temperature. Thus in winter, when the examination was made, the sweating was continuous and free. It can be discerned even in the photograph. The cranial nerves were not involved. The seventh nerve on the affected side was not paralyzed, and the muscles supplied by it responded perfectly to the

faradic current. Hearing was not acute, but it was alike on both sides. On the face, neck, trunk, arm, and leg the man distinguished accurately between heat and cold, though he winced more to heat on the unparalyzed side. The tactile and pain sensations were also apparently perceived better on the unaffected side. The examination of the eyes revealed no significant changes in the eye-grounds. The right palpebral fissure was distinctly wider than the left, the eye possibly protruded slightly (though this was doubtful), and the right pupil was at least 1 mm. larger than the left. The movements of the eyeball were good in all directions.

It thus appears that in this man the three prominent symptoms, next to his hemiplegia, were unilateral hyperidrosis of the face, dilatation of the pupil, and widening of the palpebral cleft. The patient had a history also of having had a few fits after the paralytic strokes; and about one year after his admission he went insane and was removed to the asylum.

In the abdomen, lead poisoning is supposed by some to cause irritation in the area of the splanchnic nerve, the symptoms of which are the well-known lead colic and inhibition of the action of the intestines, but this subject is still obscure.

Mills[228] reports a case of left-sided paresis, with excessive perspiration on the same side, which developed about the same time as the paresis. The secretion of the sweat glands on the right side was normal. There was also increased lacrymation on the left side and slight paresis of the muscles supplied by the seventh nerve. Occasionally the left side of the face reddened very much, and remained so for some time.

Apolant[229] reports a case of right-sided hyperidrosis of the face, limited closely to the middle line. The redness of the face was equal on the two sides; the pupils were also equal.

Guttmann[230] reports the case of a patient with moderate left-sided exophthalmus, dilatation of left pupil, enlargement of the palpebral fissure, visible perspiration only on the left side of the face and neck and limited closely by the middle line of the face, and moderate redness of the left side of the face. The left ear was especially red. This redness was present only during the perspiration. The muscles supplied by the seventh nerve were not affected, and tactile sensation was normal. The temperature of the left side of the face was increased during the abnormal perspiration. The left pupil changed in degree of dilatation under observation within a few minutes, but the change was never excessive. There was no sign of compression of the sympathetic nerves. The vasomotor signs were not always present; the oculopupillary signs were constant.

Exophthalmus is produced experimentally by irritation of the

cervical sympathetic nerves, causing contraction of Müller's fibres which are innervated by the sympathetic.

Guttmann regards his case as due to stimulation of the oculopupillary fibres and paralysis of the vasomotor, and does not think it improbable that these two conditions may exist even in lesions of the peripheral cervical sympathetic.

Pokroffsky[1] reports a case of unilateral hyperidrosis and redness of the face after eating.

Gustav Riehl[2] reports a case of recurring headache, with redness, sensation of heat, and profuse perspiration on the left side of the head, and dilatation of the pupil on the affected side. The autopsy showed round-cell infiltration and small hemorrhages in the superior cervical ganglion on the affected side. Riehl says that in by far the greater number of cases of hyperidrosis dilatation of the vessels and myosis were observed. There are cases recorded of sympathetic paralysis with myosis, vascular dilatation, and anidrosis, as was the condition in the case of Möbius.

Riehl also says that the indications of inflammation of the superior cervical ganglion in his case may show that paralysis of the vasomotor fibres and irritation of the oculopupillary fibres occurred. This is also the explanation given by Guttmann.

Eulenburg and Guttmann believe the oculopupillary fibres of the sympathetic are more peripherally situated, and are therefore more exposed to external pressure than are the presumably more centrally located vasomotor fibres. This view, however, is fanciful and unsatisfactory in the extreme. It is customary to regard the hyperidrosis as a consequence of vascular paralysis. Positive observations of sympathetic paralysis exist, in which there was myosis with reddening of the side of the face without visible perspiration, and other observations in which perspiration without redness was present.

Profuse sweating has been caused by irritation of the cervical sympathetic in animals. "The hyperæmia and dilatation of the vessels, lasting a short time, in hyperidrosis unilateralis may just as well be explained by irritation of the vasodilators as by paralysis of the vasoconstrictors. As in our case, a decided symptom of irritation — mydriasis — was associated with hyperidrosis and vascular dilatation, the explanation of all these symptoms as the result of irritation of the fibres in the sympathetic is reasonable" (Riehl).

J. Friedländer[3] says that this condition (hyperidrosis unilateralis), to which attention has only recently been paid, occurs either as part of a symptom-complex, sometimes only temporary, or as an independent, or at least predominating, phenomenon in persons otherwise

healthy and normal. The following cases, according to him, belong in the first category. Nitzelnadel (1867), by whom the first observation of this anomaly was made, reported the case of a patient, aged forty-seven, with Graves' disease, in whom the left side of the face alone was found to perspire freely. Nitzelnadel observed also sudden and profuse sweating, especially on the left side of the face, in a case of diabetes mellitus. Külz reported a case of diabetes in which the hyperidrosis was left-sided. Mickle reported two cases of general paralysis, in one of which exclusively right-sided excessive perspiration of the face lasted for years; in another hyperidrosis was due to paralysis of one side of the face, and lasted only a few weeks. Meschede reported a case of temporary dementia with unilateral sweating of the face. Wiedemeister reported a case of mental disease, in which the hyperidrosis began below the left eyelid, extended to the left cheek, forehead, and left side of the scalp, sometimes crossing the middle line. The sweating was increased by lively conversation. Morselli reported a case of a woman with glioma in the anterior part of the left hemisphere, in whom during life the right side of the face was very red and covered with perspiration; the left side was very pale.

According to Friedländer, this excessive perspiration may occur in hemiplegia. Ringer, and J. S. Bury observed two cases of hemiplegia which showed constant profuse perspiration on the affected side. Messedaglia and Lombroso likewise observed hyperidrosis paralytica sinistra in a hemiplegic young man who had formerly been epileptic; and Chevalier, from similar observations, speaks of "*hémiplégies sudorales.*"

Tabes with hyperidrosis unilateralis is mentioned in the literature: Nitzelnadel reported a case of tabes, in which the perspiration predominated on the left side, and was especially profuse on this side of the face, while the right side remained dry. Remak also reports a case of tabes with excessive perspiration after eating sour food.

Cases of unilateral anidrosis are reported by Brunner, Seguin, and others. Berger reports left-sided hyperidrosis of the face, head, and neck, not extending beyond the middle line, after slight exertion, in a person otherwise normal. Unilateral hyperidrosis in healthy persons has also been reported by Pokroffsky, Dow, Apolant, Guttmann, Keyes, and others.

Friedländer reports a case in which, in a healthy woman, hyperidrosis of the right side of the face, with redness and increase of temperature of the same side, and right-sided mydriasis, developed suddenly without apparent cause. He states that the coincidence of three so important symptoms makes it improbable that

this was a mere chance occurrence, and it is probable that these symptoms had a common source.

"We designate (with Henle)," he says, "as nervus sympathicus cervicalis the upper portion of the sympathetic cord, as far as below the inferior cervical ganglion, with its rami communicantes passing to the spinal cord. . . . We know that they (the central fibres) pass by means of the rami communicantes with the anterior and posterior spinal roots into the cord, that they ascend in the cord, and then, probably at first uncrossed, enter into combination with the vasomotor and oculopupillary centres in the medulla oblongata. From these centres (according to Budge) a group of sympathetic fibres, consisting of vasomotor fibres, after having passed through the cerebral peduncle—their peripheral course cannot be traced further—supply the vessels of the external ear, the side of the head and face (Claude Bernard), the cerebral membranes, possibly also the substance of the cerebral hemispheres (Donders and Callenfels). It is not known whether they supply only vasoconstrictors or also vasodilators, or whether they are crossed or uncrossed. The other group (oculopupillary tract), which probably does not have a common course with the vasomotor fibres, arises in the spinal cord, from which these fibres enter the sympathetic cord by means of the two lower cervical and two upper dorsal roots (Budge), and pass then—their further course is not known—into the cranial cavity, and probably to the dilator pupillæ, by means of the first branch of the fifth nerve.

"The sweat fibres—the knowledge of the anatomical localization of which we owe to Nawrocki—leave the spinal cord and enter the thoracic sympathetic cord, ascend through the ganglion stellatum to the cervical portion, apparently in the same tract with the vascular nerves (Landois), and associate themselves in the head with the branches of the fifth nerve."

This explanation by Friedländer of the course of the sympathetic fibres upwards through the cerebral peduncle, to supply the vessels of the external ear, is very obscure. How do these fibres reach the external ear after passing *upwards* through the cerebral peduncle?

It may be considered certain, says this author, that the sympathetic nerves control the perspiration, the amount of blood in the vessels, and the width of the pupil. This statement, furthermore, is confirmed by the experiments of Wagner, Müller, and G. Fischer on the heads of those just executed.

In speaking of his case, Friedländer says there were present hyperidrosis, mydriasis, but also—and therein is the incongruity of the symptoms—vascular dilatation, two symptoms of irritation, and one of paralysis of sympathetic fibres.

If it may be thought that the vasomotor fibres have a different course from the others, which is probably not true of the sweat fibres, it is possible to believe that irritation may affect one of these groups of fibres, or that one group may be paralyzed; but the theory that simultaneously a paralysis at one place and irritation at another should exist (when there is no anatomical basis for either set of fibres), is hardly to be accepted.

He offers the following possible solution of the question: The secretion of saliva is independent of the circulation of the blood, and is under the influence of specific secretory nerves; nevertheless, irritation of these nerves (the facial nerve for the submaxillary gland) is observed to cause a considerable enlargement and hyperæmia of the vessels of the glands. One is therefore forced to assume that in the seventh nerve, vasodilator fibres, in addition to secretory fibres, exist and are irritated with the others. We may have, therefore, a unilateral hyperidrosis as the result of the irritation of specific sweat fibres in the sympathetic nerves. We know that the sweat secretion is entirely independent of the vascular condition. When we see all the signs of marked local hyperæmia with hyperidrosis, we must conclude that the cause of this vascular overfilling is due to sympathetic irritation, i.e., we must assume that vasodilators are associated with vasoconstrictors in the sympathetic. Truly the assumption of vasodilators in the sympathetic is somewhat venturesome, as they do not rest on a physiological and anatomical basis in man, although they have once been demonstrated in the dog.

Friedländer says that Seguin found the ganglion cells of the sympathetic *on both sides* filled with much pigment, which is a negative result, that Morselli found on the side of the hyperidrosis a marked sclerosis and fatty degeneration of the cervical sympathetic, that Fränkel regards the cause as abnormal enlargement of the perivascular spaces in the sympathetic ganglia, and that on this idea of Fränkel Ebstein builds a very complicated explanation. The whole subject is obscure and no definite pathology is known.

Morrow[224] had a case of unilateral hyperidrosis which disappeared spontaneously. In another case the hyperidrosis was attributed by the patient to a blow received on the bridge of the nose, causing a deflected septum. A portion of the deflected septum was removed, and the operation resulted in a diminution of his catarrh and a decrease of his perspiration.

F. Windscheid [225] reports a case of unilateral hyperidrosis of the right side of the face which extended somewhat below the lower jaw to the neck and upwards to the line of the hair. The skin of the right side of the face was warmer than on the left side, mydriasis of

the right eye was present, and the seventh nerve was normal. There was severe headache on the right side of the forehead and loss of hair all over the head. No painful points over the fifth nerve were found. He states that Adamkiewicz has demonstrated that there is a physiological connection between perspiration and the seventh nerve, and he reports several cases of hyperidrosis with disease of this nerve.

Until 1890 Windscheid was able to find only one careful study of hyperidrosis of the face; this was by P. Raymond.[115] Raymond makes the following classification: (1) Hyperidrosis faciei in consequence of disease of the central nervous system; (2) hyperidrosis faciei in consequence of disease of the cervical sympathetic; (3) hyperidrosis faciei in consequence of disease of the seventh and fifth nerves; (4) reflex hyperidrosis faciei due to irritation of other nerves (nerves of taste or sensation), or due to psychical influence. Most cases belong to the second class. He reports a case of right-sided hyperidrosis with right-sided mydriasis, and also quotes a case reported by Bichat, in which hemiplegia was present with hyperidrosis of the face on the paralyzed side.

Landois and Stirling[7] conclude that the vasomotor nerves are distinct from the sweat-secretory nerves. This is shown by the fact that excessive sweating may occur when the skin is pale, as in fear and in the death agony. Special nerves control the secretion of sweat, and their stimulation causes increased secretion—as has been shown in the amputated leg of a kitten, after complete arrest of the circulation. In the normal state, however, profuse perspiration is always associated with dilatation of the blood-vessels. The secretory and vasomotor nerves seem to lie in the same nerve trunk. Adamkiewicz and Senator found that in a man suffering from abscess of the motor region of the cortex cerebri for the arm, there were spasms and perspiration in the arm. An example of vasodilator fibres is seen in the nervi erigentes. These can be excited reflexly from the sensory nerves of the penis. The psychical disturbance which accompanies anger or shame is followed by dilatation of the blood-vessels of the head, owing to stimulation of the vasodilator fibres.

It is a question whether the slight redness of the face in my case was due to paralysis of the vasoconstrictors or stimulation of the vasodilators. The opinions given make it probable that at least in some parts vasodilator fibres exist. Foster says that "when the lingual is stimulated the blood-vessels of the tongue dilate."

Some statements have been made in regard to the presence of sympathetic fibres within the brain.

Bechterew and Mislawski,[116] by irritation of the peripheral cut end of the cervical sympathetic, caused dilatation of the pupil, prom-

inence of the eyeballs, retraction of the third eyelid, and an evident secretion of tears in the corresponding eye.

By irritation of the cortex of dogs about the sigmoid gyrus, and likewise of the portion deep within the inner part of the thalamus, at the anterior part of the gray commissure, they caused secretion of tears, dilatation of the pupil, prominence of the eyeballs, and retraction of the third eyelid. They conclude that the central tract of the cervical sympathetic is to be found in the thalamus, and ascends from here to the cortex. No mention is made in these experiments of redness of the face or of perspiration.

German authorities, as a rule, ascribe the drooping of the upper eyelid in sympathetic paralysis to paralysis of Müller's fibres in the upper lid, which are innervated by the sympathetic.

Oppenheim says that he has once observed in a case of lesion in the optic thalamus the symptoms of sympathetic irritation described by Bechterew. He also says that when there are symptoms of sympathetic paralysis there may be at the same time symptoms of sympathetic irritation, but that this is very rare.

In view of the statements which have been made, let us consider the case reported by me: The man has had two attacks of hemiplegia, which came late in life. He is paralyzed on the right side, but he is not aphasic. Therefore I do not believe the lesion is cortical. The lesion must be near the internal capsule. He has no hemianopsia, which simply means that the optic fibres, which pass through the pulvinar at the extreme end of the thalamus, are not cut. He has symptoms almost exclusively of sympathetic irritation, for there is no active flushing of the face. These have existed for some time. There is no reason to think that they are due to disease of the peripheral portion of the sympathetic, and indeed it is hard to imagine a lesion that could cause at the same time irritation and paralysis at a point where the fibres are so closely associated as they are in the cervical sympathetic cord. Centrally the vascular portion seems to be somewhat separated from the ocular portion (if we may consider perspiration as well as redness vascular symptoms), as Bechterew obtained no vascular symptoms in his experiments; and we can therefore more easily imagine a lesion at a central portion of the tracts that would cause irritation and paralysis. The problem is much simplified, however, if we conclude, as seems justifiable, that the patient has no symptoms of paralysis, *i.e.*, flushing. Sympathetic symptoms in cerebral lesions are recorded by some high authorities in neurology. We know also that within the spinal cord the ocular centres are higher than the vascular centres for the cervical branch of the sympathetic cord. Forced laughter may be due to mental failure; it may,

however, be due to a lesion near the thalamus. Bechterew[m] maintains that forced laughter is not uncommon in cerebral paralysis, although seldom mentioned by neurologists. He believes the reflex arc for laughter is through the thalamus, and when the cortical influence is cut off by destruction of the fibres from the cortex to the thalamus, the restraining influence of the cortex is lost and the least irritation, external or internal, may cause convulsive laughter. The laughter in my case, however, could scarcely be called "forced" or "convulsive," and does not require such an hypothesis to explain it as the one suggested by Bechterew. It was simply an inane smile or grin, and could be explained by the man's mental state. Absence of sensation in my case is of little significance.

In the case reported by Dejerine and Mirallié of hemiatrophy of the face with unilateral syringomyelia, the ocular symptoms due to a lesion of the sympathetic were present, but the vascular symptoms were absent.

Dastre concludes from this case that it was not simply the cervical sympathetic itself that was affected. This system, according to him, is composed of fibres that are diverse in their origins and functions. Hence the symptoms differ according as the nerve trunk or the points of origin are affected. If a lesion is limited to the spinal cord below the level of the three upper dorsal nerves, it does not affect the circulation of the face, or if this is affected, it is only temporarily so, due to the fact that the face receives part of its sympathetic innervation from the trigeminal nerve, *i.e.*, from the bulb.

As the centres for these two sets of functions, ocular and vascular, occupy such different parts in the cord, it seems to indicate that these fibres of the two sets are somewhat disunited, and that paralysis of one set with irritation of the other set is possible in central lesions.

The question of the persistence of anæsthesia in cerebral lesions is one of importance.

Oppenheim says that in tumors of the central ganglia, especially in those of the optic thalamus, disturbance of sensation, pain, paræsthesia, much more frequently hemihyperæsthesia and hemianæsthesia, have been noticed. In most of the cases of Assagioli and Bonvecchiato, Pilz, Mills, Oppenheim, Dercum, Nothnagel, Masing, Kirilzew, etc., hemihyperæsthesia or hemianæsthesia was demonstrated. Just as great, perhaps even greater, is the number of cases of tumor in the optic thalamus in which every anomaly of sensation was absent. This circumstance makes it improbable that the disturbance of sensation is a direct focal symptom of new growth in the optic thalamus or in the central ganglia.

Dejerine's case has created great interest. He says that disor-

ders of sensation in hemiplegia of cortical origin are in general transitory, and the same is true of hemianæsthesia of capsular origin.

The symptoms in his case were hemiplegia of the left side, especially noticed in the superior extremity, and almost a monoplegia, with abolition in this limb of all forms of sensation, including the muscular sense; slight diminution of sensation in the lower half of the face, the left half of the trunk, and lower limb of the same side; integrity of the special senses, and persistence of the anæsthesia of the upper limb, with loss of muscular sense during five and one-half months until death. At the autopsy the lesions found consisted of cortical softening of the right hemisphere; secondary degeneration of the posterior segment of the internal capsule, of the cerebral peduncle, pons, bulb, and cord. The fillet was intact.

Ballet during his lectures in Paris in the winter of 1895, showed a case of hemiplegia in which the loss of sensation had existed for considerably over a year.

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TROPHONEUROSES.

(EXCLUDING SCLERODERMA, ACROMEGALY, AND ADIPOSIS DOLOROSA.)

BY

CHARLES K. MILLS,

PHILADELPHIA.

TROPHONEUROSES.

UNILATERAL AND LOCAL ATROPHIES AND HYPERTROPHIES.

Hemifacial Atrophy.

Synonyms, Definition, and Literature.

The synonyms of hemifacial atrophy are unilateral atrophy of the face, progressive facial hemiatrophy, neurotic or neural atrophy of the face, facial trophoneurosis, progressive laminar aplasia, and atrophy of the connective tissue of the face. Morphœa, which was formerly classed by some authorities as a form of partial facial hemiatrophy, is now usually regarded as a variety of circumscribed scleroderma. Unilateral atrophy of the face is a neurotrophic disorder, characterized by gradual wasting of the skin, subcutaneous tissue, and bones of one side of the face. The muscles are not usually implicated, but may be in rare instances. In 1886 the writer collected about sixty cases of facial hemiatrophy from the literature on the subject. Collins, in an extensive investigation of the literature in 1895, found records of one hundred and twenty-six cases. The disease was first described by Parry, in 1825. One of the most valuable of early descriptions was that by Romberg, in 1846. A number of cases have been recorded by American writers, especially during the last ten or twelve years. Bannister, in 1876, presented a complete survey of the subject up to that time.

Etiology.

Möbius has advocated the theory that the disease is due to external local infection, believing that the infection may take place in the vessels, mucous membranes, or skin. In connection with the subject of infection it should be remembered that some of the reported cases have occurred after syphilis, scarlet fever, measles, typhoid fever, and other infectious diseases. Inheritance seems to play some part. Seeligmüller, for instance, has reported one case occurring in a child nine years old, and another in the mother of this child. The affec-

tion is of more frequent occurrence in females than in males, in the proportion probably of two to one. It most frequently originates in childhood and youth, rarely developing after the prime of life, although cases have been recorded occurring at various ages before fifty. Out of twenty-five cases analyzed by the writer, only one was over the age of thirty years, and three-fourths of these cases began before the age of twenty. Exposure to cold, inflammation of the submaxillary gland, and abscess of the ear have been followed by facial hemiatrophy; and among exciting causes, falls or blows upon the face are generally given important places. Among the diseases in the course of which hemiatrophy of the face has been observed are hysteria, epilepsy, insanity, multiple sclerosis, syringomyelia, multiple exostoses of the head and face, localized and diffused scleroderma in other parts of the body, and pityriasis rubra (Collins).

Symptoms.

The disease is insidious in origin and slow of development, the appearances first noted being usually certain changes in the skin. A white or whitish spot is seen on the cheek, and other similar patches appear from time to time, these gradually changing to a darker yellowish or brown color, the first appearing being the first to show such alterations of color. Sometimes the change in color involves the entire half of the face, or may in some positions extend a short distance beyond the median line. The face may even assume a mottled appearance, as in one of the early cases reported by Romberg, in which a yellowish-gray discoloration began at the median line and extended across the lower half of the face to the angle of the lower jaw. Patches of discolorations were also present in the upper half of the face. These changes in color in circumscribed or diffused areas are due to or coincident with advancing atrophy of the skin. In the course of time, but usually not until after the lapse of years, the atrophy attains its maximum, and then the appearances remain much the same during the life of the patient. In rare instances atrophic spots, similar to those observed in the face or to the generalized atrophy of the face, are seen in other parts of the body. The atrophy of the face is in still rarer cases bilateral.

According to Möbius hemifacial atrophy sometimes advances with all the characteristics of simple atrophy of the skin, namely, an advancement with an infiltration of the marginal wall clearing behind the atrophied skin.

The hair may be thinned or changed in color on the affected side, this being shown particularly in the beard, eyebrows, and hair bor-

dering the forehead and temple. Complete absence of the hair of the eyelid from the inner canthus to the middle of the lid has been observed (Romberg).

The bones of the jaw, and especially the upper jaw, may atrophy, and even the teeth seem sometimes to undergo atrophic changes and fall out of their sockets. Atrophy of the tongue or gums may be present.

Anidrosis, or absence of perspiration, is a common symptom, and the attention of the patient or his friends is first called to the disease

FIG. 48.—Case of Hemifacial Atrophy in which the Lesion is limited to the Forehead and Upper and Lateral Parts of the Nose. (Lloyd.)

by the fact that the patient perspires on one side of the face only, or naturally on one side only, and but very little on the other. The perspiration is usually normal in other parts of the body. Diminution of lacrymal secretion and unilateral exophthalmos have been observed.

Differences in local temperature on both sides of the face have been noted by some, but have not been found by others. Bannister, for example, observed a difference of six-tenths of a degree in favor of the ear of the unatrophied side, while others have failed to discover any differences in local temperature.

Fig. 48 represents a case of hemifacial atrophy described by Lloyd, in which the disease began as a white patch near the bridge of the

nose several years before the case came under observation. This patch gradually spread until it involved the forehead and hairy scalp. The true skin was almost destroyed, fat and connective tissue disappeared, and the bone was deeply atrophied strictly on one side.

In two cases recorded by Bannister taste was involved; in one ageusia was present on the anterior half of the tongue on the affected side, in the other on the posterior third of the tongue. In the case in which taste was impaired only over the posterior third of the tongue, and the symptoms were those of a typical unilateral facial atrophy, the simplest explanation would be that of a coincident involvement of the glossopharyngeal and trigeminal nuclei or root fibres. As Bannister has suggested, the accompanying symptoms (which were headache, mental confusion, and impaired cardiac action) indicated a widespread intracranial lesion, involving the trigeminal, auditory, and glossopharyngeal nerves, including the facial and pars intermedia of Wrisberg. The latter we regard as the central portion of the chorda tympani. In the majority of reported cases taste has not been affected.

Impairment of hearing, usually slight, has been recorded. In one of my cases careful examination showed atrophy of the membranes of the tympanum.

Ophthalmoscopic appearances with the exception of slight cupping and haziness of the disc have been negative.

Trigeminal neuralgia is a not infrequent accompaniment or complication of hemifacial atrophy. In some reported cases the patients have suffered especially from frontotemporal neuralgia, or neuralgia in some other portion of the distribution of the trigeminus. Hyperæsthesia in special areas of the face and scalp has also been observed. The neuralgic attacks are usually paroxysmal, but may frequently recur or in rare cases be very persistent. The neuralgia may or may not be accompanied by twitchings of the facial muscles. Various paræsthesiæ may be present, the patient sometimes complaining of numbness, tingling, or burning sensations. Slight tactile anæsthesia has also been noted, but marked anæsthesia of any variety is uncommon.

Hemiplegia is sometimes an accompaniment of hemifacial atrophy, as it may be of atrophy of the tongue or of any other symptom or set of symptoms referable to the cranial nerves. Sometimes the hemiplegia precedes the atrophy, at others it develops at much the same time.

In a case recorded by Diller the patient had suffered from what the reporter describes as attacks of Jacksonian sensory epilepsy, although, as mentioned by Lloyd in the discussion of this case be-

fore the Philadelphia Neurological Society, the diagnosis of epilepsy might be regarded as doubtful, the symptoms being somewhat like those present in some forms of hysteroepilepsy. The relationship between these attacks and the partial facial atrophy was not easy to trace. Besides the sensory attacks just described this patient had at least one severe motor paroxysm with apoplectic accompaniments, and subsequently suffered at times with dizziness. It is possible that a meningeal growth may have been present, so situated as to cause cortical irritation and at the same time exercise a direct influence upon one of the branches of the fifth nerve.

Twitchings of the muscles supplied by the motor distribution of the fifth may be present; irregular contractions of one of the masticatory muscles on one side have been observed (Axmann and Hueter).

Marked changes of the musculature of the face are not present in hemifacial atrophy, at least the muscles supplied by the seventh nerve are not usually involved. Some atrophy of the muscles supplied by the motor branches of the fifth nerve, namely, the masseter, temporal, and pterygoid, has been noted.

In a few reported instances unilateral facial atrophy has been found in association with insanity, and especially in cases which have shown the somatic signs of degeneration. These are to be placed in the same class, in most cases at least, with the cases of arrested and aberrant development of congenital origin. Mendel and others have reported such cases.

In rare instances of true atrophy of the same type as hemifacial atrophy, the atrophic process is confined to one-half or to a certain portion of the face. Such *semilateral atrophy of the face* has been described by Blumenau—a case in which the atrophy was distinctly confined to the lower half of the face. Besides the lip, cheek, and nostril, the atrophy also extended to the half of the tongue of the affected side, to the soft palate, and to the maxillary bone. The affected half of the lip was completely hairless, the mustache growing naturally on the opposite side. The frontal region of the face was not affected. At the time of examination the patient was fifty years old. The atrophy began when he was sixteen, the first notable feature being a whitish spot upon the upper lip. The atrophic process reached its maximum five years from the onset, and had therefore remained stationary for nearly thirty years. Sensibility, the electrical reactions, and local temperature were normal on both sides. In 1885 Weir Mitchell reported a case of absence of adipose matter in the upper half of the body, both sides of the patient's face presenting a striking similarity to the affected side in cases of unilateral or local atrophy of the face.

Morbid Anatomy and Pathology.

Autopsies have been recorded in only two cases: one of these by Homen, in which a dural tumor compressed the Gasserian ganglion and the branches of the fifth nerve—anæsthesia of the region affected, and paralysis of the oculomotor nerve being present; another case by Mendel, in which examination revealed proliferative neuritis of the left trigeminal nerve, most marked in its second branch. Central atrophy of the spinal root of the fifth and partial atrophy of the substantia ferruginea were discovered. This case is of unusual value, both because of the care with which the autopsy and microscopical examinations were made, and also because the patient had been frequently studied during life. The case was a typical one, had long before the patient's death been recorded in neurological literature, and the patient had appeared at different clinics in Germany for a period of twenty-five years. The facial atrophy in this case was associated with atrophy of the left upper extremity, and the autopsy and microscopical examination showed that the left musculospiral nerve had undergone changes similar to those found in the Gasserian ganglion and the fifth nerve.

It is generally held that hemifacial atrophy is a trophoneurosis dependent upon lesion, probably irritative in character, of some portion of the fifth nerve. It is generally considered that the trophic fibres of the fifth nerve are contained in the posterior root, and therefore the disease presumably is an affection of this portion of the trigeminus. The autopsies in the cases of Mendel and Homen support this view, which is also upheld by other facts. Section of the posterior root of the trigeminus has been followed not only by unilateral atrophy of the muscles of the face, but also by atrophy of the muscles of mastication, and even by atrophy of the tongue and of the bones of the face, and by thinning and color changes in the hair. Such associated symptoms as trigeminal neuralgia point to the fifth nerve as the probable source of the disease. The trigeminal origin of the disease cannot, however, be regarded fully as established.

Operations on the Gasserian ganglion have lent only partial support to this theory. According to Krause, after Gasserian operations, trophic diseases of the skin did not as a rule follow, the majority of cases not showing the slightest dermal change of a vasomotor or trophic character even after several years. The skin of the cheek on the operated side was a trifle smoother and a little more stretched in one of his cases three and one-half years after an operation on the second branch of the fifth. In another case, on the

operated side, the skin felt a little thicker and harder than on the opposite side. Krause, however, says that he believes these slight trophic changes may be explained on the ground that they were caused by a slight pinching of the anæsthetic mucous membrane during chewing. In the second patient referred to even the eyebrows were thinner on the operated side, but he thought this might be explained by the scar of a former operation on the supraorbital nerve. He also says that herpes did not occur, and he was unable to note any change in the secretion of sweat on the operated side.

Krause contrasts these results—this general absence of trophic affections after Gasserian and pre-Gasserian operations—with the reported conditions in cases of paralysis of the trigeminus. As is well known, many records show the presence of such conditions as herpetic changes of the skin of the face, neuroparalytic keratitis, the formation of scar tissue in the skin of the cheek and the mucous membrane of the nose. He argues, therefore, that the mere loss of nerve influence cannot be sufficient to cause such disturbances, but that other factors, especially neuritis, must enter. Not one of his cases at any period after his operations showed any appearance of facial hemiatrophy. Physiological experiment has shown that section of the trigeminus in animals causes wasting of the operated side of the face.

William Aldren Turner, as the result of experimental sections of the trigeminal nerve, arrived at the conclusion that the Gasserian ganglion does not exercise any trophic influence on the cornea, and if septic organisms are excluded, the ophthalmic branch of the fifth may be safely divided or the Gasserian ganglion removed without fear of corneal lesions. According to him, so-called paralytic phenomena associated with lesion of the trigeminus are evidences of irritation and not of paralysis. Facts of this kind are corroborated by the view that the influence exercised by the fifth is not trophic, and it would seem to follow that while lesions of the trigeminus or of its nuclei or root fibres may play a part in the production of facial hemiatrophy, when they do this the lesions are irritative in character. Some of the older observers attributed hemifacial atrophy to disorders of the cervical sympathetic, and some support to this view has been given by a case recorded by Seeligmüller, in which a tumor pressing upon the cervical sympathetic led, among other symptoms, to facial hemiatrophy.

The question arises whether hemifacial atrophy is not sometimes of cortical origin. Certainly the usual type of this affection, the form which has been described in the previous pages, does not originate from cortical lesion, but it is well to bear in mind the fact that

in recent years true atrophy has been found associated with hemiplegia.

Spiller found one case of unilateral arrest of facial development in thirty-three cases of infantile hemiplegia, studied by him at the Pennsylvania Training School for Feeble-Minded Children. This case was one of left hemiplegia with paralysis of the muscles innervated by the lower branch of the seventh nerve. According to Féré the face usually participates in the hemiatrophy of infantile hemiplegia, but Spiller regards this as a somewhat surprising statement, he having found only one case of arrested development in which the face was involved. I have seen several cases of facial arrest in infantile hemiplegics, and there is now one such case in the wards for nervous diseases of the Philadelphia Hospital. Some atrophy of the face is sometimes seen in cases of adult hemiplegia, as I have learned from my own investigations on hemiplegias, moderate or slight atrophy being not infrequently present in the limbs as well as in the muscles of the face. The distal portions of the limbs are particularly likely to suffer, the small muscles of the hands and fingers showing decided wasting, and the same is true of the muscles of the feet and lower portion of the leg. Savill has suggested that cortical trophic centres may be situated in the limbic lobe, basing this suggestion upon one or two clinicopathological observations. In rare cases partial hemifacial atrophy might be attributed to cerebral and even to cortical lesions, but such cases could be readily distinguished from those of the usual type.

Diagnosis.

The diagnosis is to be made by a careful study of the time and method of onset, history, and present condition of the patient. The disease most frequently begins in youth. The circumscribed thinning of the skin and areas of discoloration should be specially borne in mind. Careful study will show that the atrophy affects the skin and subcutaneous tissues, and possibly the bones, gums, and other structures which have been enumerated, but not the muscles; or if the musculature is at all affected, it will be so only to a limited extent, and the atrophy is usually confined to the muscles supplied by the motor distribution of the fifth nerve.

According to Möbius it is almost impossible at times to distinguish between hemifacial atrophy and scleroderma, which doubtless have close pathogenetic relations. In hemiatrophy of the face all the tissues are affected primarily, while in circumscribed scleroderma the atrophy of the deepest tissues is secondary to pressure. In morphœa,

which is closely related to hemifacial atrophy, the bones are not affected.

Congenital asymmetry of the face is commonly met with in association with aberrant or arrested development of other portions of the body, and cases of this kind are seen in institutions for the idiotic and feeble-minded. In these, face, trunk, and limbs are frequently atrophied together. The association of other somatic and of psychical evidences of arrested development or degeneracy will render clear the diagnosis of these cases from ordinary hemiatrophy. Atrophy of one-half of the cranial vault may be present, and this is most likely to be associated with atrophy of the face, trunk, and limbs of the opposite side.

In very rare cases, in individuals otherwise normal, one side of the face is considerably smaller than the other. These cases sometimes occur in persons of average intelligence and mental development, but in them the special signs of unilateral atrophy will be absent.

A case of hemifacial atrophy may for a time through carelessness be regarded as facial paralysis. In the former the muscles supplied by the seventh nerve are not invaded; willed movements of all kinds can be readily performed; the muscles respond promptly to the electrical current; while in peripheral facial paralysis the opposite is true. It would be only in extremely rare cases, in which some atrophy of the face is present in association with hemiplegia, that the diagnosis could remain even for a brief time in doubt. No one who fully comprehends the diagnostic features of that very common disease, Bell's palsy, could long be mistaken in his study of a case of facial hemiatrophy.

In a special type of progressive muscular atrophy the muscles of the face are first implicated. In progressive muscular atrophy the appearances of the skin shown in cases of facial hemiatrophy are not present, and the subcutaneous tissues and bones and the other structures affected in facial atrophy are not involved. As a rule in facial hemiatrophy the atrophic processes spread over very nearly the entire half of the face, although, as has been indicated, we occasionally have cases of semilateral atrophy, but even these cases show a certain regularity in the distribution of the atrophy and a tendency to follow the course of subdivisions of the fifth nerve. The order and method of progress of a case of progressive muscular atrophy are very different from those of facial hemiatrophy.

A glance at a patient suffering from hemihypertrophy may at first sight lead to the opinion that the face of the unaffected side is atrophied, but careful scrutiny will, of course, soon correct this error.

Prognosis.

No case of recovery from hemifacial atrophy is on record. The disease does not, however, progress all through life, the atrophy as a rule reaching its full development in a few years. The case studied by Virchow and Mendel, and on which an autopsy was made by Mendel, lived for from thirty to thirty-five years after the inception of the disease.

Treatment.

In the cases so far reported no treatment has proved of any permanent value. Möbius has suggested as prophylactic treatment that surgical interference over the infected area might be of benefit. Sachs has recommended thyroid feeding, which, so far as I know, has not been given a thorough trial. The application of galvanism to the affected tissue seems sometimes temporarily to stay the progress of the affection. Dercum has suggested early resection of the branches of the trifacial, believing that this treatment would do good on the assumption that the disease is due to perversion of the trophic nerve stimulus, but no results have been recorded as following surgical measures of this kind. Massage may be tried in the early stages, but it should not be too vigorous, and care should be taken not to cause excoriation of the skin by the treatment. This may result because of the lowered resistance of the atrophied parts. Measures designed to improve the general health of the patient should be employed, as in most atrophic diseases this tends at times to be affected. As some disfiguration of the face results from the wasting, a light rubber plate or pad can be introduced into the mouth and attached to the patient's upper teeth, this being made of such size and contour as to fill out the cheek and give it a more nearly normal appearance (Sachs).

Hemilingual Atrophy.

Hemilingual atrophy or hemiatrophy of the tongue is a wasting confined to one-half of the tongue. As an isolated affection it is very rare. It is usually observed in connection with chronic degenerative diseases. Dr. C. W. Burr has recently reported an interesting case of hemiatrophy of the tongue, and has reviewed the literature of the subject.

Etiology.

The disease occurs most frequently between adolescence and old age, although it may appear at any period of life. Syphilis may be ranked among the most frequent predisposing causes, and other forms

of infection may play a part. It has also been attributed to the effects of lead and other toxic agents. The general etiology of lingual hemiatrophy is indeed practically the same as that of hemifacial atrophy, with the exception that the former more frequently develops during mature life. When an isolated affection, it is, according to Hirt, usually due to traumatism, disease of the vertebral artery, new growths in the oblongata, or embolic softening in the region of the hypoglossal nucleus.

Pathology and Morbid Anatomy.

When hemilingual atrophy is an isolated affection the lesion is most probably situated in the nerve trunk, although this does not

Fig. 49.—Hemilingual Atrophy in a Case of Syringomyelia.

necessarily follow. Degeneration in rare cases attacks only the hypoglossal nucleus, and it is conceivable that a small macroscopic focal lesion may be limited to the nucleus or root fibres of this nerve. A small tumor or a localized meningitis may be so situated as to involve the hypoglossal, spinal accessory, and pneumogastric nerves as they

pass from the brain stem in close proximity to each other, or the glossopharyngeal and other adjacent nerves may be implicated. It is nevertheless true that the evidences are in favor of the lesion being

FIG. 50.—Hemilingual Atrophy in a Case of Degeneration of Various Cervical and Bulbar Nuclei.

situated within the oblongata when various other nerve distributions are affected conjointly with the hypoglossal.

The lesions that may be found in hemiatrophy of the tongue, as enumerated by Burr, are chronic nuclear degeneration, especially in association with disease of the spinal cord; cysts and softening the result of hemorrhage, embolism, or thrombosis; a tumor arising

within the postoblongata, and confined to one side; an exostosis or a tumor growing from the membranes of the pons; hydatids, caries or meningitis, or direct injury from wounds. It is frequently associated with tabes, and may occur in the spinal type of general paralysis, in syringomyelia, and in disseminated sclerosis, and may be a part of the syndrome of bulbar paralysis. In Figs. 49 and 50, two examples of lingual hemiatrophy are shown, the first in a case of syringomyelia, and the second in the case of a man who presented symptoms indicating degeneration of various cervical and bulbar nuclei. When associated with the chronic degenerative diseases, the lesion is probably primarily one affecting the ganglion cells of the nucleus of origin of the nerve. In not a few cases it is probable that an acute apoplectiform attack due to embolism, thrombosis, or hemorrhage is the starting-point of the affection.

Symptoms.

In hemilingual atrophy one side of the tongue appears considerably smaller than the other, the amount of diminution depending of course upon the extent and stage of the disease. The tongue has a tendency to go to one side on protrusion, the side to which it turns being that affected by atrophy, because of the action of the muscles of the opposite side, this action being of a pushing and protruding character. Fibrillary twitchings are sometimes, but not invariably, present in the atrophied muscles; similar contractions may be present upon the unaffected side. Speech, swallowing, and mastication may be affected through implication of either the nerves or the muscles.

Bilateral atrophy of the tongue is occasionally observed, particularly in connection with progressive muscular atrophy and progressive bulbar paralysis, and more rarely in tabes.

In a case seen by Dr. Rugh and the writer, the patient, a woman twenty-two years old, was first seized with a severe pain in the right scapula. The attack of pain, which was pronounced to be rheumatic, lasted three weeks. She soon noticed that the scapula was becoming prominent, and later began to suffer from weakness in her right arm. Close examination showed atrophy of the rhomboid, the supraspinatus, infraspinatus, and deltoid muscles on the right side, and the same on the left, but to a less degree. The muscles of the left half of the face were also distinctly atrophied, the mouth and nose being somewhat drawn to the right. The general bulk of the tongue was diminished, its right half being relatively much more atrophied than the left. Voluntary movements of the tongue were

weakened, and fibrillary tremors were present. Involvement of the ocular muscles was evidently beginning to take place. In looking both outwards and inwards the excursion of the left eye was incomplete; and in looking upwards it had a tendency to wander outwards. Some restriction of movement inward was also observable in the right eye. It is probable that the impairment was of conjugate ocular movement to the left. The pupils responded to light and accommodation. Touch, pain, and thermal senses were all preserved. Parts of the body other than those above alluded to did not seem to be involved in the atrophy.

Diagnosis, Prognosis, and Treatment.

The *diagnosis* can, as a rule, be readily made by a careful examination of the organ. Hemiglossitis is one of the affections from which it must be differentiated, but the symptoms of this disorder are of an active character, and are readily recognized. The tongue, like any other portion of the body, may be congenitally smaller upon one side than the other, but this is infrequent, and would not be accompanied by the symptoms which indicate loss of power in the tongue. Hemihypertrophy of the tongue is exceedingly rare, but its presence might at first lead to the idea that the unaffected side of the tongue was atrophied.

The diagnosis of the pathological nature of the affection and the focal diagnosis are sometimes important. Fibrillary twitching is in favor of nuclear disease, but is not absolutely pathognomonic, as twitching has been recorded as present in peripheral disease. The well-known facts regarding secondary twitching and contractures as late symptoms in uncured or partially cured cases of facial paralysis due to peripheral neuritis, indicate that in some cases of lingual atrophy, even though twitchings and contractures are present, the disease may have been of peripheral origin. Perhaps the distinction might be made in these cases from true fibrillary twitchings by the larger and coarser character of those which are the secondary results of a peripheral neuritis.

The localization of the lesion causing the lingual hemiatrophy, as well as the diagnosis of its nature, may be largely assisted by a study of the accompanying symptoms. When hemiplegia of the side of the body opposite to the atrophied half of the tongue is present, the lesion is probably gross, and of such size and character as to involve the pyramidal tracts before their decussation. Sensory and other symptoms are likely to be present in such cases because of the involvement of the important tracts and centres which are associated in this section

of the neuraxis. When scattered but considerable portions of the musculature of the body are affected conjointly with atrophy of the tongue, the lesion is probably one of the evidences of a disseminated or diffused degeneration. Nuclear ophthalmoplegia of a more or less complete type may be present in such cases.

The *prognosis* is almost uniformly unfavorable.

The little *treatment* available is in the main the same as that used for hemifacial atrophy. Electrical treatment has been recommended in the peripheral forms of the disease.

Hemifacial Hypertrophy.

Unilateral hypertrophy of the face, or facial hemi-hypertrophy, is a rare trophic disease, characterized by enlargement of the tissues of one side of the face, in its most complete form involving skin, subcutaneous tissue, bone, and other structures. The literature of the subject is scanty, one of the most interesting cases being that of Montgomery.

Etiology and Pathogenesis.

Most of the reported cases of facial hemihypertrophy have been of congenital origin, and were associated with other aberrations of development. In a case recorded by Dana, unilateral hypertrophy of the face was one of the features of gigantism. In the case reported by Montgomery the development of the affection was preceded by abscess of the cheek, this coming on shortly before the first evidences of hypertrophy. In acquired cases it seems to be pretty well established that irritation of the fifth nerve, either at its origin or in its course, may play some part. In several instances it has developed during or after the course of inveterate trigeminal neuralgia. It is not improbable that in some cases of facial hemihypertrophy irritative lesions implicating the substantia ferruginea and descending root of the fifth nerve, or other peripheral or central portions of the trigeminal apparatus, may be the active pathological causes of the tissue overgrowth. It has been suggested that the affection may be of vascular origin, due to a chronic or recurrent hyperæmia, but it is more probable that the abnormal vascular states are secondary to the abnormal conditions of the nervous centres and tracts. Post-mortem investigations have not as yet yielded anything of value bearing upon the nature of the disease.

In Montgomery's case a piece of skin from the malar region and another from the chin were removed, and were examined microscopically. The sections were found to be extremely brittle, easily break-

ing along the sebaceous glands. The skin in the vicinity of these glands was lacking in resistance, as shown on introducing sutures into the face. After operation all of the specimens removed showed considerable round-cell infiltration along the course of the blood-vessels. Little tumor-like elevations of the skin were found to be composed of masses of concentrically arranged epithelial cells which had undergone hyaline degeneration. Montgomery concluded that the hypertrophy of the soft parts was to be referred to an increase in the size of the hairs and of the sebaceous glands, also to plugging of the sebaceous glands, and probably also to an increase of the connective tissue and fat. The sebaceous glands were enormously increased, especially in the cheeks.

Symptoms.

In Montgomery's case * the disease was first noticed at the age of ten years. It began in the gums and was almost exactly confined to the left of the median line, although the upper lip and gum of the right side were slightly encroached upon. The limitations of the hypertrophy were almost exactly those of the distribution of the fifth nerve. The deformity was included in an "irregular quadrilateral figure, bounded by a line starting on the top of the head at the anterior extremity of the sagittal suture, running down the nose to the lower border of the chin; then from the chin upwards and backwards, along the lower border of the inferior maxillary to the tip of the left mastoid process, and thence upwards to the point of commencement, the anterior extremity of the sagittal suture."

The most marked enlargement and deformity may be shown in particular portions of the head and face, as in the supraorbital, malar, and mental regions. In Montgomery's case both soft tissues and bones were affected by the abnormal process, but in the case of Dana the soft tissues escaped. It will be remembered that in Dana's case the facial hypertrophy was a part of a general process.

The face sometimes has a swarthy or saddle-leather look. The mouths of the sebaceous follicles are enlarged, the hair is coarse, and the lanugo or down on the nose and ear may become as large and stiff as ordinary hair. Enlargement of the ear was present in Montgomery's case, and was caused by hypertrophy of the cartilage. The comedones are unusually numerous. Bony enlargements above and about the orbit give the forehead and eye an extremely abnormal

* The signs and symptoms of this affection, at least when it is acquired, cannot be better given than by an enumeration of the salient points of Montgomery's description.

appearance. The orbit and eye may present a beetling, cavernous look.

The gums and alveolar processes may show an overgrowth. In several of the reported cases the alveolar processes were enlarged. The facial expression is changed, but paralysis of the facial muscles is not present, the patient usually having full control over willed movements of the face.

In Dana's case of gigantism and acromegaly, the left side of the face showed remarkable osseous hypertrophy, involving the frontal bone and the upper and lower jaws, corresponding closely with the distribution of the left trigeminal nerve. The enlargement gave a curious twisted and asymmetrical look to the face. In this case, as in others, the appearance of the face at first gave the impression of atrophy of the comparatively normal side.

In Montgomery's case the tongue was not hypertrophied, and the vessels were normal, but in several cases recorded by others the vessels were unilaterally hypertrophied.

Increased flow of saliva has been noted, and increase of perspiration on the side affected, especially in congenital cases.

General sensibility and the special senses are not usually affected, although in a case recorded by Friedreich sight and taste were both diminished.

The field of vision may be blurred by the faulty removal of the lacrymal secretion, faulty because of the deformed upper lid. In Montogmery's case the discs were excavated on the left or affected side more than on the right, but optic neuritis was not present, and there were no signs of intracranial pressure.

Diagnosis, Prognosis, and Treatment.

The diagnosis must, in the first place, be made from hemifacial atrophy, for, as already stated, in some of the cases the appearances give rise at first to the impression that the other side of the face is wasted. Close inspection of the different tissues and structures of the face, and of ocular, facial, and mandibular movements will soon clear up this point. It is possible, but not probable, that some acute inflammatory affection of the face, jaw, or skull might need to be differentiated; but an inflammation of the gums or cheek, an abscess, chronic periostitis from syphilis and similar affections, could be readily excluded on careful examination. As the cases are usually congenital, and the result of arrested, aberrant or abnormal development, the prognosis is of course unfavorable. Even in the acquired cases, so far as the meagre records go, little can be hoped for

in the way of improvement or cure. The course of the disease is usually a slowly progressive one. In rare instances compression of the brain or of important nerve structures may result from the augmentation of the osseous and other tissues. In the recorded cases no treatment has proved of avail.

Hypertrophy of One-Half of the Body.

Occasionally cases are observed in which, at birth, one-half of the body or one limb is much larger than the other, and as a rule the proportionate difference in size between the two sides of the body is preserved as the child continues to grow. Möbius has reported a remarkable case of this kind, in which the right lower extremity was of enormous size as compared with the left, and the whole right half of the body was hypertrophied. In most cases of this kind the skin and subcutaneous tissues are the seat of greatest change. The bones are implicated in a fair percentage of cases, but the muscles only in rare instances. While the local hypertrophy is generally confined to one side of the body, this is not always the case; it may be present in several areas on both sides, and Friedreich has reported one case in which the right lower extremity and the left face and left arm were hypertrophied. The pathogenesis of these curious cases of local hypertrophy has not yet been clearly determined. The few facts which are known with regard to facial atrophies and hypertrophies suggest the view that in some, if not in most of the cases, neural irritation and degeneration play the most important rôle.

Localized Atrophies and Hypertrophies.

A considerable number of localized atrophies and hypertrophies, that is, of atrophies and hypertrophies limited to special portions of the body, have been described. Sometimes these cases are congenital, sometimes they are apparently of infectious or traumatic origin. They include cases of linear atrophy, atrophy or hypertrophy of the fingers or toes, hypertrophy of one or more extremities, and other special varieties of abnormal increase or decrease of the body tissues. Sir Dyce Duckworth has recorded a case in which the patient, three months after recovering from a severe attack of enteric fever, complained of extreme sensitiveness over the region of the thighs and over the malleoli, and examination of the parts revealed several longitudinal spots of linear atrophy and hyperæsthesia. This case, as the author indicates, points to the fact that both trophic and sensory branches of the nerves were affected by the special toxin of the fever, neuritis with neural atrophy probably having been present.

Other cases of localized linear atrophy have been recorded as occurring after infectious fevers.

Hyperostosis of the Cranium.

Hyperostosis of the cranium (*hyperostosis cranii*) is a disease, usually of trophic developmental origin, in which the bones of the cranium are hypertrophied, the enlargement extending over a large portion of the skull or being confined to limited areas.

Among the terms which have been used to describe hyperostosis of the cranium or conditions closely allied to it, are leontiasis ossea (Virchow), and megalocephalie (Starr). By some writers it has been regarded as identical with the osteitis deformans of Paget, but it is questionable whether the cases properly classed as hyperostosis cranii belong in the same category with Paget's osteitis deformans, a disease which manifests itself chiefly in the long bones, and comes on usually after middle life. Virchow suggested the name leontiasis ossea because it seemed to him that the osseous hypertrophy was analogous in nature to elephantiasis, which affects the skin and soft tissues.

History and Literature.

By far the most valuable contribution to the subject of hyperostosis of the cranium is an article by J. J. Putnam, of Boston, who has not only recorded observations on five personal cases with an additional case of Morton Prince, but has carefully reviewed the literature and theories of the disease.

Prior to 1892 only one paper of importance on the subject of hyperostosis cranii seems to have been published, and that was by Virchow. Baumgarten, in 1892, analyzed the clinical history of such cases as he could discover in medical literature, and described thirteen famous skulls showing hyperostosis, found in the museums of Europe. Le Dentu, in 1879, described hyperostosis chiefly as it occurs in disease of the jaws. Putnam gives a brief bibliography of the subject at the end of his article, and refers in the body of it to several American contributions. Besides the papers of Virchow and Baumgarten, one of the most important is by Poisson, who suggested the theory of trophoneurosis to explain the disease. He described ten specimens, and analyzed or summarized about thirteen clinical observations, but some of the cases referred to by him certainly did not belong properly to the disease under consideration. M. Allen Starr, in 1894, published an account of a case for which he suggested the name megalocephalie. Edes, in 1896 (in the same number of the

American Journal of the Medical Sciences in which Putnam's paper appears), records a case with two illustrations. Hinsdale, in 1896, published the description of a skull showing hyperostosis of the cranium, the specimen being in the Mütter Museum of the College of Physicians of Philadelphia, to which it was presented by Dr. Charles Herwisch. Diller, of Pittsburg, has also recently recorded a case.

Etiology and Pathogenesis.

A few facts regarding the general etiology of this disease are known. It usually originates in early life; of fourteen cases to which reference is made by Putnam, in seven it was initiated under the age of seven years. Of those over the age of seven, in one the age was between twenty and thirty, and the other six were over thirty. The disease does not often begin after middle life, and its typical form undoubtedly begins in youth. It is of more frequent occurrence in females.

The disease has been attributed to injury of the head in a small number of cases, but the real influence of traumatism may be regarded as doubtful since injuries are of frequent occurrence, and the tendency is to attribute all forms of acquired local deformities to traumatisms.

With regard to the pathogenesis of hyperostosis cranii, the weight of opinion seems to be in favor of its trophic or developmental origin. In its typical forms it can probably be regarded as a true trophoneurosis. Virchow, however, held that localized osteophytes and general hypertrophies of the bone, such as occur in hyperostosis cranii, were due to local processes of irritation or inflammation—a view which has been upheld by others, and which is probably correct for some cases of bony enlargement, but not for those which are usually classed as hyperostosis cranii. Even Virchow regarded heredity as a probable predisposing influence, and also believed that the disease was more likely to occur at certain developmental periods.

Starr regarded the disease as of trophic origin like acromegaly, and in support of this view called attention to the associated thickenings and enlargements in the neck, skin, and in the features. Putnam also refers to the fact that in some of the cases changes had taken place in the neck and in other parts of the body of a character which indicated that some general condition was at the bottom of the disease.

While many facts and analogies would seem to support the trophic theory, most of the pathological observations which have been made in special cases seem to be opposed to it. With regard to this point Putnam speaks as follows:

"As against the trophic theory is the fact, which for the present must count for the essential one, that the pathological change hitherto observed seems to be an inflammatory one, involving the periosteum, the bone, the dura mater, in varying degrees, and usually leading to an obliteration of the diploë. Sometimes the diploë is rarefied, to

FIG. 51.—Hyperostosis of the Cranium. (Hinsdale.)

be sure; but oftener it is changed to an ivory-like tissue, such as is occasionally seen in syphilitic skulls. In acromegaly, on the contrary, the changes are, on the whole, rather of the nature of hypertrophy, and signs of periostitis are, to say the least, not prominent."

SYMPTOMS.

The hyperostosis may predominate in one of at least three places, namely, the cranial vault, the bones of the upper face, or the lower jaw. In the recorded cases the disease has been manifested most markedly and frequently in the upper face. The appearance of the head and of

the features changes to a remarkable degree, as the disease progresses, and as is strikingly seen in some of the photographic illustrations of the disease at different stages.

Among the symptoms or affections which have been observed as

Fig. 52.—Hyperostosis of the Cranium. (Hinsdale.)

occurring at any period in the history of hyperostosis cranii, are epilepsy, erysipelas, suppuration of the ear, and suppuration of the tear duct.

Among symptoms directly attributable to the pathological process are pain in the head, which may be either localized or diffused, pains in the limbs, exophthalmos, mental irritability, drowsiness, loss of hearing, taste, or smell, and optic neuritis with secondary atrophy. Insecurity in walking and awkwardness in the use of the hands have also been noted, and are directly or indirectly to be referred to the disease. The headache, or head pains, have been found in about half the cases which have been recorded, and these pains as a rule

have appeared to be osteoscopic, that is, due to the irritative process going on in the bone itself, and not to pressure. Optic neuritis was observed in one of Putnam's cases and in the case recorded by Edes, while atrophy of the optic nerves was present in Prince's case, and was probably consecutive to a neuritis which had been present before the case came under the observation of Prince. Loss of teeth has been observed in several cases. The hair sometimes becomes coarser.

Diagnosis, Prognosis, and Treatment.

In the early stages of hyperostosis cranii it is possible that the pain, usually more or less localized, might lead to the supposition that a neuritis or ordinary periostitis was present. As the disease advances, the only affection with which it is likely to be confounded is intracranial tumor, and indeed the conditions which are present within the skull are similar to those which we have in brain tumor; in other words, when circumscribed enlargements of the bones are present on the outside of the skull, endostoses may be present on its inner aspect, and Virchow long ago suggested what is now well known to be the fact, that such internal osseous thickenings are likely to occur at points within the cranium correlated to cranial exostoses. The fact that papillitis and atrophy have been observed in a few cases also makes the diagnosis from brain tumor of more importance. The diagnosis is of course to be made by a study of the progress of the case, and especially of the data which indicate that the bones of the jaws, face, or cranial vault have gradually hypertrophied. While pain in the head is of frequent occurrence, it is usually not of the agonizing character observed in cases of brain tumor. The local paralyses and spasms and the affection of the special senses due to local intracranial processes are not present.

The progress of the disease is slow. As stated it usually begins in youth, and the patient may live until middle age or beyond it. The prognosis is unfavorable, although in some of the cases the disease has remained stationary for a considerable period. No treatment in recorded cases has proved of avail. Putnam has suggested trephining when the symptoms of intracranial pressure and irritation are severe and the external affection is somewhat circumscribed. Owing to the thickness of the skull, however, such an operation would be tedious and difficult. It might be worth while to try the operation simply for the purpose of relieving pain and retarding the progress of the disease. Putnam has also suggested that the process of osseous hypertrophy might be retarded by cutting off the blood supply of the bone. In one of Putnam's cases the throbbing in the head,

congestion of the face and sweating, which distressed the patient, were relieved by pressure made by a light steel spring and pad which was so applied as to compress one of the carotid arteries, and the question of ligating the carotid or one or more of the meningeal arteries is worthy of consideration. Thyroid treatment might be tried.

RAYNAUD'S DISEASE.

Synonyms, Definition, and Literature.

Raynaud's disease is a vasomotor and trophic affection, paroxysmal in its most marked manifestations, usually bilateral and symmetrical, and characterized by such phenomena as pallor, coldness, numbness, pains, anæsthesia, and gangrene, attacking by preference the fingers and hands, although the toes and feet, and even the ears, nose, and other parts may be affected.

In 1862 Maurice Raynaud carefully described the affection, which in its most complete type is characterized by local gangrene of the parts affected. Frequently the disease presents itself in several stages, to which special names have been applied, and hence, although it has numerous synonyms, none of these is fully applicable to all phases of the affection. In one stage of the disease the chief manifestations are those of *local syncope*, and the affection, so far as observation extends, may not pass beyond this condition, which is characterized by extreme coldness with a shrinkage or contraction of the tissues. In other cases *local asphyxia* is primarily developed, the parts becoming swollen, turgid, and livid, showing extreme stagnation of the entire capillary circulation. The disease in some instances may not pass beyond this stage. When local asphyxia is present only in the extremities, the affection is sometimes designated *acroasphyxia*, and at other times as *dead fingers* or *dead toes* (*digiti mortui*). The last stage of the disease is usually that of *local gangrene*, the affection then being known by various names, such as *symmetrical gangrene of the extremities, local gangrene of nervous origin, sphaceloderma, gangrene of the skin*, and *neuropathic gangrene*. The disease usually shows itself in the extremities as a symmetrical affection. In rare instances the nose is affected. Two of the stages or varieties of the disease may appear in the same subject and even in adjacent regions; some parts, for example, appearing as cold and cyanotic, while others are swollen, red, and hot.

In Volume V. of the present work, Leloir, in an article on "Dermatoneuroses," has described this disease at some length, but some of the neurological features of the affection are not there considered.

For a full description of some of the most important phenomena of the disease at different stages, the article of Leloir should be consulted, as they will not be here repeated in detail.

The literature of the subject of Raynaud's disease is now very large. Among early publications of importance on this and allied subjects, after the papers of Raynaud, were those of Billroth, Weir Mitchell, Allan McLane Hamilton, J. C. Warren, Weiss, R. Lauer, Schulz, and Lutz. These and other writers are referred to by M. Allen Starr in Pepper's "American System of Medicine," 1886, and since this date the literature of the subject has very rapidly expanded. The writer in 1878 reported an interesting case of Raynaud's disease in the *American Journal of the Medical Sciences*. Among important articles of comparatively recent date are those of Wiglesworth, Affleck, Collier, Collins, Henry, and Osler.

Etiology.

The disease occurs more frequently in the female than in the male sex, in the proportion, perhaps, of two or three cases to one. It is rare beyond the age of fifty, and most frequently appears between the ages of twenty and fifty, although typical cases have been observed in children. Exhausting diseases and any causes or conditions which lead to impoverishment of blood and weakened nerve power may predispose to the affection, which has been observed in the anæmic, neurasthenic, hysterical, and those suffering from such constitutional affections as syphilis, tuberculosis, and cancer. Occupation may be a predisposing factor. In some instances it is an exciting cause, as when the disease occurs among laundresses or washerwomen whose duties require them to have their hands for a long time in water or other liquids of different temperatures. A few cases have been attributed to injury. Some cases have been ascribed to blows or falls so received as probably to affect the solar plexus or special ganglia of the system of gangliated nerves, but these cases seem to be of doubtful authenticity. Some authors, and among them Raynaud, have attributed considerable influence in the production of the disease to disorders of menstruation, but the majority of writers seem to doubt the importance of the part played by this process. Among exciting causes fright and emotional disturbances of various kinds should be given an important place, and special occupations and local exposures to heat and cold may have some influence. The disease has also been observed among alcoholics and among patients who are habituated to the use of opium and other narcotics. It is believed by some to be most frequently due to toxic causes. It follows or is associated with

infectious diseases in some instances. It may be a concomitant or complication of other organic diseases, particularly those of a chronic and degenerative character, such as tabes, syringomyelia, myelitis, and bulbar paralysis, and it has not infrequently been observed among the insane.

Leloir calls attention to the coëxistence of the asphyxial form of the disease with tabes, and also to the occurrence of the affection in connection with intermittent fever, referring to several writers who have reported such cases, and to three cases of his own.

Pathology.

Raynaud ascribed the disease to irritation or increased action of the excitomotor portion of the spinal cord, believing that the gangrene was dependent upon some lack or perversion of innervation of the capillaries. It will therefore be seen that Raynaud held to its central origin. According to another view the condition is due directly to peripheral irritation. Different symptoms or stages have been attributed to different actions upon the vasomotor nervous system—the stage of local syncope to irritation of the vasoconstrictors and that of local erythema and asphyxia to irritation of the vasodilators. Another view held by a considerable number is that the affection is of reflex origin, and it is by this mechanism that menstrual disorders have been supposed to be efficient in its causation. The well-determined fact that exposure of the affected parts to cold or wet or to alternations of cold and heat may sometimes bring on paroxysms of the disease, would seem to indicate that in some instances at least it may be of peripheral origin. On the other hand, the effects of sudden emotion point to central disturbance or to general disturbance of the entire nervous apparatus. Doubtless the mechanism is different in different cases.

Henry has carefully summarized and discussed the different theories of the cause of Raynaud's disease, which are (1) that it is due to endarteritis obliterans; (2) that it is due to peripheral neuritis; and (3) that it is the result of vascular spasm. With this writer I believe that the theory of arteriole spasm is certainly the one that is most in accordance with the clinical phenomena, and this was the theory to which Raynaud gave his adherence. "The disease is most prevalent in females and in the young, *i.e.*, in those whose vasomotor system is most impressionable. It occurs in paroxysms which are caused by the surest exciter of vascular spasm—cold." Finally, in several cases during the paroxysm there has been dimness of vision, which was shown by the ophthalmoscope to depend upon a contraction of the central

artery of the retina and its branches, and in one recently reported by H. M. Thomas the attacks of local syncope were followed by a chill, loss of consciousness, and convulsions. Such facts are in the highest degree corroborative of Raynaud's view, that the disease known by his name is due to an "enormous exaggeration of the excitomotor energy of the gray parts of the spinal cord which control the vasomotor innervation."

"The symptom hæmoglobinuria, occasionally observed, is best explained by the theory of vascular spasm. The origin of the hæmoglobinuria may be twofold: it may be due to excretion of hæmoglobin that has been separated from the red corpuscles in the peripheral asphyxiated parts—nose, ears, and fingers—or it may be due to an asphyxia of the renal or other internal vessels."

Endarteritis obliterans can be excluded as a cause of genuine Raynaud's disease, although occasional cases somewhat closely resembling this affection may be due to arteriosclerosis. Such cases have been recorded by Jacoby, and are cited by Henry, one in a boy who developed nephritis with chronic albuminuria and died of apoplexy, and another in a syphilitic subject. Against the theory of endarteritis obliterans are the early ages of many of the patients, the paroxysmal character of the symptoms, and the comparative rarity of Raynaud's disease. According to Henry, neuritis, when present, is probably of secondary origin. My own experience would lead me to coincide with him in this view.

Symptoms.

The symptoms of Raynaud's disease differ according as they are observed in the stage of local syncope, local asphyxia, or local gangrene. These symptoms, in accordance with these stages, are so fully given in the article by Leloir that it will not be necessary to repeat them here, but reference will be made to some points in the symptomatology not fully described by this author. The most important symptoms when the disease presents itself as local syncope are pallor, which may become extreme, with coldness and contraction of the tissues. Sensibility is diminished, especially the tactile sense, and pain is almost constant.

The chief phenomena when the affection presents itself first as local asphyxia are lividity from capillary stasis, with, in some instances, swelling and increase of temperature. In others cyanosis and mottling of the skin are present; pain is persistent and often severe, and the skin may be anæsthetic. When the disease passes on to the gangrenous stage the skin becomes dark and insensible, and sometimes necroses in spots. The disease differs from true gangrene

in that the gangrenous spots disappear, perhaps to reappear in the same place or near by.

It has been shown that intermittent hæmoglobinuria may occur during a paroxysm of Raynaud's disease, or, to all appearances may be a substitutional condition, taking the place of the attack.

Numerous nervous symptoms have been described as accompanying Raynaud's disease, ranging all the way from simple depression or a sense of nervous weakness, to coma and convulsions. Disturbances of all the special senses have been noted, and especially visual and ocular affections. In some instances the visual fields are contracted or obscured, and internal ophthalmoplegia has been observed.

Osler, after referring to the frequency with which Raynaud's disease is met with in forms of insanity, says that in a few cases cerebral manifestations, due apparently to vascular changes similar to those which develop in the peripheral parts, have been described. In the case of a man in whom epileptic attacks occurred in the winter months only, in connection with local asphyxia and superficial necrosis of the ears, the patient had also hæmoglobinuria. In another case, that of a woman aged fifty-two, local syncope and asphyxia occurred at intervals during a period of six years in the fingers and hand of the right side, sometimes with aphasia, and on several occasions with transient paralysis of the right arm and leg. In the final attack the patient died with gangrene of the right hand and arm. Weiss has reported another case in which aphasia complicated the disease. In a third case "falling attacks" of an indefinite character occurred in a young girl, with local asphyxia from the knees to the ankles.

Diagnosis.

The diagnosis of Raynaud's disease is usually not difficult if moderate care is taken by the investigator. The affections with which it is possible to confound it are gangrene due to cardiac disease or to occlusion of the vessels from embolism or endarteritis, and the gangrene associated with diabetes and nephritis. Erythromelalgia resembles some of the stages of Raynaud's disease. The eponym, Raynaud's disease, should be restricted to cases in which the disease is a true trophoneurosis. Besides the forms of gangrene just enumerated as not belonging to the class of Raynaud's disease, other varieties, usually of symmetrical type, may result from such causes as severe infection, disturbances of hæmatopoiesis, and ergotism. In connection with the careful etiological study of the case, the bilateral symmetry of the affection, its mode of onset and progress, and the absence of evidences of disease outside of the neural apparatus will serve to

make the diagnosis clear. Lévi has recorded cases of what he regards as hysterical forms of Raynaud's disease, and of erythromelalgia. One case of this disease he reports as having occurred in an hysterical and hypnotizable subject, who presented also abnormalities of the urinary functions. He believes that the hysterical form of Raynaud's disease may be established by a powerful emotion, and by the transformation of this affection into a subconscious imperative emotion, the disease becoming an entity. The onset is sudden and of emotional origin, with associated renal disease, either anuria or polyuria. In hysterical cases the disease is presented as a local syncope or asphyxia, although Lévi believes it may be possible for gangrene to result. The attacks are temporary, but may frequently recur, and, according to Lévi, are distinctly influenced by hypnotic or post-hypnotic suggestion. The question of the existence of an hysterical form of Raynaud's disease is of course of importance in the diagnosis of the affection.

Legroux raises the question whether chilblains, local asphyxia, and symmetrical gangrene may not represent different degrees of a necropathic dystrophy, of which the most striking example is furnished by syringomyelia. Chilblains do not come without special exposure to cold, and while cold may determine an attack of Raynaud's disease or increase its violence, this affection may appear without relation to changes in temperature. Legroux regards predisposition as essential to the production of chilblains.

The symptomatology of erythromelalgia clearly separates it from Raynaud's disease. This symptomatology in one of its most usual forms is well described in Weir Mitchell's own words: "The patient, nearly always a man, after some constitutional disease, like a low fever, or after prolonged physical exertion afoot, begins to suffer with pain in the foot or feet; usually it comes in the ball of the foot, or of the great toe, or in the heel; and from these parts it extends so as to involve a large portion or all of the sole, and to reach the dorsum, and even the leg. More often it is felt finally in a limited region of one or both soles, and does not extend beyond these areas. At first it is felt only towards night, and is eased by the night's rest; but, soon or late, it comes nearer and nearer to the hour of rising from bed. In like manner, while at first it is made to increase only by excessive exertion afoot, by and by it comes on whenever the upright posture is assumed, or even when the foot is allowed to hang down. Since, however, the disease is not necessarily progressive, there are instances in which the pain never passes a definite limit. One case may for years have the trouble only in the evening; a second may reach and remain at the point where only a long walk in summer

causes it; a third may stand still, as it were, in a far more advanced stage of the malady, and, though suffering horrible pain, become no worse; while in the gravest cases, more familiar signs of organic disease of the spinal cord may arise to shed light upon the pathology of the minor forms of the trouble."

The symptomatology of erythromelalgia is varied and modified in different cases, but preserves its general characteristics as exhibited in the above description. The patients use different terms as descriptive of the pain experienced, but in the vast majority of cases it is spoken of in terms which indicate its "burning" character. This pain may be light and moderate, or of a severe and even torturing character. The horizontal position and cold relieve the pain. The patients are usually better in cold than in warm weather. Changes take place in the appearance of the feet. The skin in the parts most affected, for instance, may come to assume a dusky, mottled-red appearance. Pressure and standing or walking may increase the pain, tenderness, redness, and congestion of the parts. The flushing may come on rapidly or slowly, and involves both arteries and veins. In the most severe cases the patients are unable to stand. The cases are rarely curable, but may be improved by rest, cold, and general tonic treatment. In some instances they gradually become worse and worse.

Prognosis.

The prognosis of Raynaud's disease must be considered from two points of view: (1) As to fatality in paroxysms of the disease, and (2) as to the influence of the disease in shortening life. In regard to special attacks of local syncope or local asphyxia, or even of symmetrical gangrene, the prognosis can be regarded as good. The cases do not end fatally, and in most instances the affected parts recover, to a certain degree at least, their local functions, although it may be to have future syncopal, asphyxial, or gangrenous attacks. The general health of the patient suffering from Raynaud's disease is not, as a rule, good; some of its victims, as in one case of my own, die from phthisis, and others from diabetes. As has been indicated, they may be sufferers from degenerative diseases, such as posterior sclerosis, syringomyelia, or progressive muscular atrophy. The prognosis as to recovery from the attacks will depend largely upon their severity and the extent of the tissues involved. The disease sometimes precedes scleroderma of the fingers or sclerodactylia. Starr has recorded a case which seems to show that the disease can be entirely outgrown. He had a little girl under observation for three years, she having been first seen at the age of three years. Ten years later she pre-

sented no signs of the disease. The cases improve in the warmer weather, and some of the milder type may recover when they are no longer exposed to the cold.

Treatment.

The indications for treatment are (1) to improve the general health of the patient; (2) to improve local circulation and nutrition; and (3) to relieve the pain which so often accompanies the local disorder. Strychnine, arsenic, manganese, iron, quinine, cod-liver oil, malt, and light wines all have some value in improving general health, and wherever possible change of air and surroundings should be had. Sea voyages are advantageous, and a change from cold to warmer latitudes during the winter may prove of benefit. The extremities should be kept warm; the parts, for instance, can be wrapped in wadding. Electricity, both faradism and galvanism, has been recommended, and the latter is especially worthy of trial in the early stages of the disease. A method which has much in its favor is that of applying the current by placing the hands of the patient in vessels containing warm water through which the galvanic current is passed. Massage with oils or ointments, and both passive and duplicated movements of the extremities are measures of treatment which have much to recommend them. Nitroglycerin has been suggested with the view of affecting the circulation of the extremities. To relieve pain in extreme cases anodynes, often codeine or morphine, may be required, although it is not well to resort to these drugs because of the danger of establishing the habit for them.

When the history of a case shows that syphilis, either inherited or acquired, is present, antisyphilitic treatment should be employed. Several cases of Raynaud's disease with such histories have been put on record. In these cases the affection may be explained either on the theory of a syphilitic arteritis or of angiospasm due to a toxin. An organic lesion of the brain, spinal cord, or abdominal cavity should always be diligently sought before applying treatment (Eskridge).

PERFORATING ULCER OF THE FOOT.

Perforating ulcer of the foot (*mal perforant du pied*) in rare instances is an isolated affection, but commonly it is one of the local trophic manifestations of certain degenerative and inflammatory diseases of both the central and the peripheral portions of the nervous system. It long ago received the attention of Vulpian, and was early studied also by Duplay and Morat. It is an ulcerative process, as a rule developing slowly, and located usually somewhere on the plantar

AINHUM.

Ainhum is a trophoneurosis which usually results in a spontaneous amputation of the little toe, although in exceptional cases other toes and even the fingers may be affected. In one reported case the disease is recorded as having progressed as far as the middle third of the leg. An interesting summary of the facts known regarding ainhum is presented in a paper by Dr. Walter L. Pyle, published in 1894.

In Brazil the disease is known as *ainham*, or *quigila*, and in India as *sukha pakla*. Other names which have been given to this disease, in obedience to various hypotheses of its nature, are *amputating leprosy*, *pityriasis Ethiopium*, and *scleroderma annulare*; it is also known as *dactylolysis spontanea*.

The first report of a case was made by da Silva Lima, of Bahia, Brazil, in 1866. Numerous cases have been reported since, covering almost every part of the globe—Brazil, Egypt, Trinidad, the African coast, Algiers, Madagascar, India, China, Ceylon, British Guiana, the West Indies, Polynesia, and various parts of the United States and Canada. Up to the time of the publication of Dr. Pyle's paper the following cases had been seen in this country: Three cases in West Virginia in three negroes in the same family; a case in a negress from North Carolina; cases in Philadelphia and in Canada in negroes with North Carolina antecedents; a case at Darlington, South Carolina; five cases in Louisiana, and one case in a negro, fifty years old, seen by Dr. Pyle in Washington, D. C.

Etiology.

The peculiarities of climate and soil are supposed to have some influence in its development, but the facts are not decisive, and indeed are somewhat conflicting. According to some it is most common in damp and rainy weather, while others report cases from the dry highlands of India. Sandy soil and working in salt have been assigned as causes. It can only be said that it is a trophoneurosis of uncertain origin. Local irritation is another assigned cause; thus it has been attributed to sand or other particles irritating the furrow in which the disease usually begins, to the wearing of rings on the toes and to what is called "stone bruise." The theory that the affection is due to pathogenic organisms has been advanced but has not yet been proved.

The influence of heredity seemed to be exhibited in a case which occurred in the practice of Dr. George B. Simpson, of Weston, W

Perforating ulcer has been noted as the result of disease or injury of various nerves, and especially of the sciatic. In one recorded case a perforating ulcer of the foot formed after the wounding of the sciatic nerve by a ball, and in another the same lesion occurred from compression of the sciatic at its origin by a cyst of the sacrum. Among chronic degenerative diseases those in which it has been noted are tabes, disseminated sclerosis, Friedreich's disease, progressive muscular atrophy, syringomyelia, and paretic dementia. It has been observed in anterior poliomyelitis, and it also occurs in the various forms of neuritis, as in those which are caused by toxic agents such as lead, alcohol, mercury, and arsenic, and those which follow infectious fevers. Any disorder causing a profound nutritive change may produce ulcerative perforations.

The dorsal spinal ganglia are from analogy the most probable seats of the initial lesion. In many cases it has been attributed to disease of the spinal cord, but it is not probable that this is the primary seat of the affection. It is now known that the primary lesions of locomotor ataxia are in the dorsal spinal ganglia.

The only recorded pathological appearances are those which have been noted in connection with autopsies and microscopical examinations in cases of tabes and other chronic degenerative diseases.

The existence of a local lesion, like perforating ulcer, should always lead to an examination of the patient for evidences of a more general disorder, such as tabes or syringomyelia. The disease is often bilateral and more or less symmetrical, or a fully formed ulcer may be present on one foot while a callosity and other evidences of perforating disease occur on the other foot. Althaus records a case of tabes in which areas of soreness in the soles of both feet gradually turned into perforating ulcers, this being the first symptom.

The *prognosis* when perforating ulcer occurs in connection with degenerative disease is unfavorable. The ulcers may heal under proper treatment; in some instances amputation of a toe becomes necessary, or even a portion of the foot will need to be removed. When healing occurs, relapses are comparatively common, especially if the patient attempts to stand or walk. When the disease is purely local the ulcers sometimes heal and remain healed.

Treatment.—Patients, in the first place, should be taken off their feet, complete rest of the affected parts being essential. Antiseptic dressings and applications of bovinine have proved useful in some cases. In the Philadelphia Hospital the sores are washed out with a solution of mercuric bichloride and peroxide of hydrogen, and then bovinine is applied directly or by injection.

This process of furrowing never advances in soldiers, and has been given a name more expressive than elegant. In ainhum the toe will in a few days swell, and a pain, burning or shooting in nature, may be experienced in the foot and leg affected. Pain, however, is not constant. There may be an erythematous eruption accompanying the swelling. The furrow increases laterally and in depth, and meets on the dorsal aspect of the toe, giving the toe the appearance of being constricted by a piece of fine cord. As the furrow deepens the distal end of the toe becomes ovoid, and soon an appearance as of a marble attached to the toe by a fibrous pedicle presents itself. By this time the swelling, if any, has subsided. The distal end of the toe bends under the foot and becomes twisted when walking, and causes inconvenience, and, unfortunately says Pyle, it is in this last stage only that the patient presents himself. There is in the majority of cases a small ulcer in or near the digito-plantar fold, which causes most of the pain, particularly when walked on. The ulcer does not occur early and is not constant. The case under my observation showed no ulceration, and was absolutely painless, the negress applying for diagnosis rather than treatment. The furrow deepens until spontaneous amputation takes place, which rarely occurs, the patient generally hastening the process by his own operation or by seeking surgical treatment. A dry scab forms at the furrow, and when picked and repicked constantly reforms, being composed of horny desquamation or necrosis."

The disease is not usually symmetrical or simultaneous in different toes. Cases with anæsthesia have been reported, although as a rule sensation is not abolished. Careful histological studies of the changes which occur in the parts affected have been made, especially by Eyles. The conclusions reached by Pyle are as follows: "In ainhum there is, first, simple hypertrophy, then active hyperplasia. The papillæ are pushed down and deprived of blood supply and undergo horny change. Meanwhile the pressure thus exerted on the nervi vasorum sets up vascular changes which bring about epithelial changes in more distant areas, the process advancing anteriorly, that is, in the direction of the arteries. This makes the cause, according to Eyles, an inflammatory and trophic phenomenon due mainly to changes following pressure on the vasomotor nerves." It is probable that changes take place in the nerve cells and fibres, but these have not been demonstrated. The disease would seem to belong to the class in which are included anæsthetic leprosy and Morvan's disease.

"The *diagnosis* is easy. No other disease resembles it. Gangrene is distinctly different. In frost-bite there is a history of cold, and this affection is of short duration. The absence of constitutional

symptoms and its limited field of occurrence distinguish ainhum from leprosy and from elephantiasis."

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TROPHONEUROSES

(SCLERODERMA, ACROMEGALY, AND ADIPOSIS DOLOROSA).

BY

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PHILADELPHIA.

SCLERODERMA, ACROMEGALY, AND ADIPOSIS DOLOROSA.

SCLERODERMA.

SCLERODERMA is a disease in which the connective-tissue elements of the skin, either with or without a previous period of œdema or infiltration, undergo sclerosis or atrophy. As we will presently see, this definition is insufficient, inasmuch as both clinical and pathological facts indicate that the connective-tissue elements of structures other than the skin may also suffer. Indeed, so extensive is this involvement at times, including muscles, bones, joints, and mucous membranes, that to define it as a disease of the mesoblast better accords with the clinical findings. Again, the presence at one time or another of either localized or general nervous phenomena suggests that the definition of the affection must be still further enlarged so as to include the nervous system, and it is because of the presence of these nervous phenomena, though they are admittedly variable, that the disease is here considered among the trophic affections.

HISTORICAL SKETCH.*

There can be no doubt that the affection was known in some degree to the ancients. Various descriptions given by Hippocrates, Galen, Avicenna, Paul of Ægina, and others suggest very strongly the scleroderma of to-day. It is not, however, until the middle of the seventeenth century that a description appeared in medical literature which is unmistakably that of scleroderma. We refer, namely, to that given by Lusitanus of a woman in whom the skin became very hard and the entire surface of the body assumed the appearance in thickness and resistance of leather. Subsequently similar descriptions were given by Diemerbroeck and also by Helvetius. In the eighteenth century Martin, Vater, Curzio, and Marteau also described cases clearly referable to scleroderma. The descriptions of Currie and Haller must also be included as must also that of Lorry. In the

* In the historical sketch of the affection, Lewin and Heller's treatise on "Scleroderma," Berlin, 1895, has been freely used.

early part of the present century cases were described by Alibert, Strambio, Casanova, Henke, Baerman, Pierquin, Fantanetti and Froriep. The first accurate description of the affection, however, was given in 1845 by Thirial, who, in addition, endeavored to establish the identity of the affection he described with that of sclerema of the new-born. The name scleroderma we owe to Gintrac. To Ball we owe the first detailed description of sclerodactyle.

Various names other than scleroderma have been proposed; for instance, sclerema or pachydermatous disease, elephantiasis sclerosa, sclerosis corii, trophoneurosis disseminata, etc. However, the name scleroderma is now by common consent universally used. Numerous papers upon the subject have appeared of recent years and gradually our conceptions of the disease have become more and more extended, so that the circumscribed affection is considered as identical in nature with the diffuse affection; so also sclerodactyle is regarded as but a local expression of the general pathological process.

Etiology.

Inasmuch as scleroderma is quite a rare affection, it has been difficult to establish the various factors which play a rôle in its causation. However, in the admirable collection of cases made by Lewin and Heller, some four hundred and seventy-five in number, certain facts become evident. In the first place, it must be considered as established that women are affected far more frequently than men, indeed in a proportion somewhat greater than that of two to one. It is a remarkable fact also that certain local symptoms, such as sclerodactyle, are very rare in men.

By far the greater number of cases occur between twenty and forty years of age. This statement, however, needs some qualification as regards women, in whom the process tends to begin somewhat earlier than in men, a relatively large number beginning before the age of twenty years. Exceptionally in either sex, it begins before the age of ten. A comparatively small number of cases appears between forty and fifty and the number steadily diminishes as old age is approached. This is especially true of men in whom cases of scleroderma beginning after fifty are excessively rare. In women, on the other hand, scleroderma after fifty, though infrequent, is much more often seen than in men. Only three cases are on record as having begun after seventy.

Regarding race, it may be said that no nationality is exempt. The greater number of the recorded cases are either of German or French nationality, a much smaller proportion being either English or American. In Italy and Spain as well as in Slavic and other coun-

tries the number of recorded cases is very small. In considering these facts, however, we must remember that they are in part to be explained by lack of recognition and lack of study of the disease in many localities.

With regard to occupation, no definite statements can be made, although now and then occupations accompanied by exposure to cold appear to favor scleroderma. Heredity, on the other hand, plays absolutely no rôle. This is also true of infectious diseases. Among diseases which occasionally precede scleroderma and possibly bear some etiological relation to the latter, we must mention functional nervous troubles, more especially hysteria and neurasthenia.

While we are very much in the dark regarding the real nature of scleroderma, it is well recognized that among the exciting causes three factors play a more or less marked rôle. It is, for instance, not an uncommon thing for the patient to attribute the onset of the disease to fright or other sudden psychic shock. Again, it is not uncommon for the disease to follow a severe trauma, as for instance a severe blow upon the head; it may be that here nervous shock is again the real factor at work. In another group of cases we find the disease attributed to exposure and to cold. All of these factors have been present in cases studied by the writer. Thus in one patient the scleroderma was attributed to the fright caused by a fire. In another it followed a blow upon the head, the blow being of such force and character as to produce a severe lacerated wound of the scalp over the occiput and to be attended by unconsciousness. In a third case again, the disease was attributed by the patient to the fact that she had for years been in the habit of entering a small ice-house many times a day; only those portions of the surface not protected by clothing were involved, namely, the face and neck and the hands and forearms as far as the elbows. Another patient attributed her disease to the fact that she persisted in washing clothes in the open air during bitterly cold weather, and in this instance it was the face, neck, forearms, hands, and feet which were involved. In a very large number of cases, however, no history of any exciting cause can be elicited.

Symptoms.

The symptomatology of scleroderma varies greatly not only with the extent of the disease but also with the stage at which a given case may be studied. We have present as the most prominent symptom a change in the true skin. This change may be attended in its early periods by an infiltration of the connective-tissue elements. The skin under these circumstances may become indurated and hard or it may

become much swollen and œdematous. On the other hand, infiltration may be absent or so slight as not to attract attention. In such instances the changes are apparently at first those of simple and very gradual atrophy. In cases in which the skin has been first infiltrated or œdematous atrophic changes also ensue at a later period, due in all probability to the reabsorption of an exudate. These differences in the mode of the involvement of the skin suggest a classification of cases convenient for purposes of description. Thus we recognize first an *œdematous form;* secondly an *indurated form*, that is, a form in which the skin without being œdematous or swollen is merely indurated, and thirdly a *form without infiltration.* Scleroderma is further separated clinically according to the extent and distribution of the symptoms. Thus we recognize especially a diffuse and a circumscribed form. It may involve more or less the entire body or it may be limited to definite areas. Sometimes it occurs in very small patches. These small patches have been described by dermatologists as a separate disease under the name of morphœa, but there is every reason for considering morphœa as identical with scleroderma.

FIG. 54.—Diffuse Scleroderma. Stage of infiltration.

In discussing the detailed symptomatology of scleroderma it will be most convenient to consider first that of the diffuse form. As already stated this may be either œdematous, indurated, or non-indurated. If the case be one in which œdema is present in the early stage, we will notice that the skin of some portion of the body, more frequently of the hands, is slightly swollen and stiff. The patient, for instance, will notice that a ring fits tighter upon the finger than before, and soon the sense of constriction may be so great as to lead the wearer to remove the ring altogether. Then as a rule the patient realizes for the first time that the fingers and hands are distinctly swollen.

The swelling may extend slightly above the wrists or to a variable extent up the forearm and arm. At the same time it is noticed also that the face is slightly swollen. The œdema of the face is very different, however, from that which is noted in Bright's disease, the swelling being decidedly more resistant. Little by little this œdema becomes more marked, more widely diffuse, and gradually approaches in character an induration so that the skin feels hard and firm. Motion then becomes slightly interfered with at the joints, more especially in the fingers and hands. Although this form is termed œdematous because of the decided swelling of the skin, it requires as a rule firm and persistent pressure to cause the swelling to disperse, and even then well-marked pitting can only with difficulty be produced. As the infiltration becomes more marked the face becomes rounded and more or less distended and the features obtunded. The feel of the skin is characteristic during the height of the swelling, being so tense and firm that it is not possible to gather it into folds. It has evidently lost its elasticity and to some extent even its mobility over the subjacent tissues. The diffuse swelling, as already stated, may involve the entire trunk as well as the arms and legs. Occasionally, though rarely, it is found that in some one area the infiltration is more marked than elsewhere. Thus in a case studied by the writer, while diffuse infiltration was found all over the body, a special mass or swelling existed over the anterior aspect of the thigh just above the knee, and this swelling pitted slightly upon pressure. During the height of the infiltration the patient's face may bear a superficial resemblance to that of one with myxœdema, but the examination of the swelling by the finger at once dispels the illusion. In myxœdema the skin feels soft and almost jelly-like. In scleroderma it is more or less hard and resistant. The characteristic changes, too, noted in the hair in myxœdema and the broadening of the fingers and nails are also absent in scleroderma. In color the skin is whiter than normal, the flesh tints being absent. Sometimes it is so white as to resemble marble. Later on, as the infiltration persists, various pigmentations of the skin may appear. Brownish or yellowish patches, small or widely diffused, may make their appearance over the chest, abdomen, buttocks, back, or arms; more rarely upon the face. Sometimes this pigmentation is very extensive and may assume a gray, greenish, or reddish hue. In one of the writer's cases it was very marked upon the abdomen, the latter being of an almost uniform greenish-brown tinge. In other cases again the pigmentation is very slight and may be limited to oval or rounded patches of violaceous coloration in the axillæ and groins.

At the time that these changes are taking place in the skin, pain

is often noted in the joints. This pain bears no relation to the degree of infiltration of the surrounding skin. It resembles in character the pain of ordinary rheumatism. It is as a rule made worse by movement. More or less marked neuralgic pains may also make their appearance. At times they are limited to definite nerve trunks.

FIG. 55.—Scleroderma. Stage of atrophy and sclerodactyle.

These symptoms of pain may and frequently do persist throughout the entire course of the affection.

After the swelling has persisted for a number of months, possibly for a year and even longer, it is observed that the degree of infiltration of the skin becomes less. Little by little the swelling of the features becomes less pronounced, the cheeks become slightly flattened, and the nose slightly sharpened, and at the same time that these changes are apparent in the face, similar ones are noticed over the trunk and limbs. In other words, after the stage of infiltration and œdema has persisted for some time a stage of resolution or, as commonly expressed, of atrophy sets in. Gradually the swollen and distended skin becomes less and less indurated, but as it does so it

tends to contract and in this way produce characteristic changes in the features and other portions of the body. If atrophic changes become pronounced, the skin is tightly drawn over the features, which acquire a mask-like expression. In well-marked cases the features are so stiff and leather-like that a change of expression on the part of the patient is almost impossible. Indeed, frequently the patient can open the mouth only with difficulty, rarely to the full extent. The sharpening and narrowing of the nose already noted become more and more pronounced until its shape is much changed. The mouth becomes drawn so as to look more or less puckered. The ears also may show signs of shrinkage and distortion. The chin, too, may become sharpened very much as is the nose. The eyes are apt to present a curious appearance due to the narrowing of the palpebral fissures. In some instances, however, the palpebral fissures may be wider than usual, the patient being unable to entirely close the eyes. The changes noted in the skin of the face are also noted elsewhere, and soon the patient finds that the movements of the arms and legs are much restricted. Thus the arms cannot be raised as easily or abducted from the body as freely as before. Flexion and extension also become somewhat limited. If the scleroderma has involved the legs the atrophic changes produce more or less interference with movement in walking. The stride may be much shortened, movement at the knee and ankle joints may become much restricted, and the entire limb on either side may be stiffened and rigid. Under these circumstances a patient may be unable to mount a stairway or may do so with great difficulty or perhaps only by using one foot at a time. Any attempt to extend the limbs beyond the limit to which they are voluntarily moved by the patient produces pain. At this stage of the disease the patient also complains of more or less marked sense of tightness and constriction about the face, limbs, and joints.

It is in the hands, however, that the most interesting changes occur. In by far the larger number of cases of scleroderma occurring in men the hands and feet escape, but this is not true of women. In women the infiltration or atrophic change in the skin is accompanied by more or less marked fixation of the fingers and thumbs. As a rule the fingers are fixed in a position of semiflexion. Sometimes they are markedly contracted; at other times, again, they are grotesquely distorted. As a rule the skin at this stage feels hard and dense. In color it is white and to the touch it is very cold. Indeed the hand often looks and feels very much like the hand of a marble statue, but the color may vary from time to time, being sometimes slightly more tinted with pink. Pigmentation also may be noted, more especially about the finger tips and nails. At times, especially after ex-

posure to cold, the fingers may become cyanosed, the condition then suggesting Raynaud's disease. Scleroderma of the hands with its attendant fixation and contracture is termed technically sclerodactyle. As already stated, it occurs almost exclusively in women; two exceedingly well-marked cases, however, have been observed by the writer in men.

As the sclerodactyle becomes more and more pronounced the fingers begin to lose their full and rounded contour. They become at

FIG. 56.—From a case of Scleroderma, showing the Hand in Sclerodactyle.

times pointed, owing to the absorption and shrinkage of the pulp of the tips of the fingers; at other times they become somewhat club-shaped. At times, also, marked atrophic changes take place in the bones. Sometimes entire phalanges of the fingers may be destroyed. Frequently it is one or more of the distal phalanges that suffer, though at times it is the proximal phalanx or the middle phalanx that disappears. If it is the distal, it is apt to be accompanied with permanent destruction of the nail and matrix and shortening of the fingers. The skin gradually becomes more and more tightly drawn over the knuckles and over the phalangeal joints so that the latter can almost be studied through the atrophied skin. That the nutrition of such a skin should be excessively low can be readily understood. Slight blows upon the knuckles are apt to cause sloughing and persistent ulceration, conditions which disappear or heal with very great difficulty.

Just as infiltration and fixation of the hand and fingers may take place, so infiltration of the foot and toes may occur. Fixation, distortion, or contracture may be observed in them just as in the hands.

As in the case of the hands, the feet are as white as marble and the toes are rigid. Pigmentation appears to be somewhat less common than in the hands.

While these changes are taking place in the skin various changes may occur elsewhere. Thus, in addition to the pigmentation already mentioned, patches of erythema may make their appearance in various situations, remain for a time, and again disappear. So, also, telangiectatic patches may occur upon various portions of the body, more especially upon the face. In these telangiectatic patches, dilated small blood-vessels can, as a rule, be readily distinguished, and the face may look reddened and the skin irritated and angry. As a rule patches of telangiectasis come and go very much as do the patches of erythema. In other instances, however, they become more or less permanent, thus adding to the unsightliness of the face.

Occasionally ulcers make their appearance. These may be situated in various parts of the body. Sometimes, as in a case observed by the writer in which an ulcer formed over the right occipital region, the lesion is accompanied by excessive neuralgia. Sometimes an intense burning pain is felt in the ulcer itself. These pains are as a rule controlled with great difficulty. The ulcers themselves heal very slowly. In some cases small vesicles may make their appearance; in others pustules are noted. Sometimes, again, appearances resembling urticaria are seen; at other times patches resembling ecchymoses. Other trophic changes have also been noted, as, for instance, herpes zoster. Changes have also been observed in the hair; sometimes the hair of the scalp becomes thin or dry and brittle; sometimes circumscribed loss of hair takes place. In the nails changes may also be noted; these structures may become ridged, thin or thickened, and sometimes brittle.

One of the most striking changes observed in the atrophic processes of scleroderma is the loss of subcutaneous fat, and this may add greatly to the change observed in the countenance. As a rule the fat is lost above and below the zygomatic arch and above and below the clavicle, and the patient may appear excessively thin and emaciated. The loss of fatty tissue frequently involves the entire body so that the arms and legs partake of the same wasted appearance as does the face and neck. The trunk also may seem excessively emaciated so that merely a thin atrophic skin is interposed between the finger and the ribs. Further, contractures may steadily progress until marked fixation takes place in various portions of the body; thus the movement of the neck may become much impaired, the power of flexing the trunk may be very largely lost, while the fixation of the limbs may become so marked that it is impossible for

the patient to walk, to dress, or even to feed himself. A patient may remain in this condition not only for months but for many years, or death may ensue due to some intercurrent affection. Sometimes the difficulty in taking and assimilating food becomes so great that the patient succumbs to a gradual process of inanition. In other cases it is a bronchitis which is the cause of death, the chest walls being so fixed that free expectoration becomes impossible. Other causes, such as pneumonia, intercurrent heart or lung affection, or accidental sepsis through the infection of a trophic lesion may terminate the case. The fixation of the joints is not entirely dependent upon the degree of change in the overlying skin. In fact there is sometimes marked interference with movement when the degree of change in the skin is very slight. For instance, in a patient under the care of the writer the fixation of the jaw is so marked that the teeth cannot be separated by a greater interval than an eighth of an inch, yet the skin of the face and cheeks is quite movable.

It must not be inferred from the description given that scleroderma is a disease which necessarily pursues the above course. It may be spontaneously arrested in any stage. Again, resolution may be so marked and so complete that the skin assumes a practically normal condition and in such instances true recovery may be stated to have taken place. In an analysis made by Lewin and Heller of two hundred and fifty-one cases, forty, or a little less than one-sixth, recovered; in seventy-five improvement was noted; no change or increase of the disease was noted in seventy-four, and in sixty-two death occurred.

In the indurated form of the disease, that is in which there is merely a board-like induration of the skin without true swelling, the progress is in every other respect identical with that of the œdematous form, fixation, contractures, and the various trophic changes which we have already described taking place. In the third form, namely, in which there is neither œdema nor induration, the skin passes spontaneously into the condition of contracture and fixation. It is probable, however, that even in this instance some infiltration precedes the atrophic change because more or less hardening of the skin is noted in the areas which are contracted; otherwise the symptoms pursue a course in every way similar to that above described.

At various stages in the course of scleroderma nervous symptoms may be noted. We have purposely avoided their mention for fear of confusing the clinical picture. It will suffice to say that nervous phenomena of one kind or another are almost invariably present. It is true, however, that they vary greatly in character,—that they are alike in no two cases. Thus we may find hypochondriasis, hysteria,

mental depression, melancholia, and even mental impairment; or we may find more positive symptoms, such as insomnia, giddiness, headache, and neuralgia. Pains referable not only to the joints but also to various nerve trunks and closely resembling neuritis may be observed. In other cases, again, chorea, tremor, ataxic movements, spasms of muscles, fibrillary twitching, or local paresis may be noted. Sometimes, again, symptoms suggesting intracranial lesion, such as inequality of the pupils, may be present. In very many cases, again, the patient complains of disturbances of sensation, such as paræsthesia. Only rarely has anæsthesia been noted. A sense of tightness and constriction independent of the condition of the skin has also been observed. The great variety of the nervous phenomena would indicate that they are of an unessential character, and yet the fact that nervous symptoms of one kind or another, although limited in number, are usually present, indicates that the nervous system is more or less involved in this curious disease. As showing the inconstant character of the nervous phenomena, we need but refer to the reflexes as exemplified in the knee jerks, which may be normal, exaggerated, or diminished.

In addition to the various phenomena presented by the skin and the various symptoms indicative of nervous derangement, special symptoms may be present referable to the digestive tract, the circulatory apparatus, the kidneys, and more rarely to the lungs. Thus it is not an uncommon experience to meet with more or less marked gastro-intestinal atony. Occasionally a gastric catarrh is met with. Frequently a tendency to diarrhœa is observed. At times diarrhœa comes on in sudden attacks and may be quite profuse. It is apparently not related to the character of the food that is taken or to such extraneous circumstances as the taking of cold. It appears to be a diarrhœa of nervous origin.

On the side of the circulation, we occasionally note increased rapidity of the pulse and sometimes well-marked attacks of cardiac palpitation; pericarditis, though rarely, has also been observed. It is difficult to assign to these symptoms their proper value. It would seem almost as if they were due to some incidental affection. As regards the urine, interesting facts are occasionally recorded. Slight albuminuria, glycosuria, and phosphaturia have been noted in a few instances. These conditions, like the heart changes just alluded to, are evidently without clear significance, especially as in the large majority of cases albuminuria and glycosuria are not observed. Phosphaturia appears to be more frequent than either of the other conditions. An interesting symptom which was first observed by M. Kohn, is that of a diminution in the output of urea. This obser-

vation has been confirmed by the writer, who determined a very decided diminution in the output of urea in two cases. In one instance less than two hundred and seventy-five grains were excreted daily, and in the other less than two hundred grains. It should be stated, however, that both of these cases were in an advanced atrophic state of the disease, and, irrespective of special pathological conditions, we must be careful not to attach too much importance to these findings, for the influence of food, of exercise, and of other factors influencing tissue metabolism must be taken into account. We must remember that patients in the advanced or atrophic stage of the disease consume but little nourishment, take almost no exercise, while the general body weight is also much diminished. In a case studied by the writer in the early or œdematous stage of the disease, the output of urea was found to be somewhat increased instead of diminished. In two cases the phosphates were found to be in excess; in another they were about normal in amount. In none of the cases was the uric acid appreciably changed in quantity.

The amount of urine appears to be frequently diminished, especially in the advanced stages of the affection. Several times irritability of the bladder has been noted, leading to markedly increased frequency of micturition.

Such phenomena as are presented by the function of respiration appear to depend upon the rigidity of the skin and muscles of the chest. Thus dyspnœa has been noted in a number of cases, as has also diminished lung capacity. That phthisis should have been noted under these circumstances would of course not appear surprising, and yet such a complication is exceedingly rare. The examination of the blood occasionally reveals interesting findings. In a series of examinations made by Dr. A. E. Taylor in two cases of the writer, the leucocytes taken as a whole were much in excess. In one of the cases albumose, a substance not before detected in the blood in scleroderma, was discovered. In another instance peptone was found in the urine. Oro found an increase of eosinophile cells. This was not observed in the cases studied by Heller. Franke, again, noted a small increase in the eosinophile cells. The observations thus far made are of course insufficient to enable us to draw conclusions in regard to the blood changes. Such changes as have been noted simply indicate some gross derangement of nutrition—of tissue metabolism. This, for instance, must be the significance of the presence of albumose, to which, however, no special significance can be attached as the observation stands alone.

On the part of the lymphatic system it is interesting to observe that occasionally the lymphatic glands are found somewhat enlarged.

Symptoms referable to the special senses are, as a rule, not present. Neither vision, hearing, taste, nor smell is interfered with. Very rarely, as has been already stated, disturbances of cutaneous sensibility are observed. These consist of numbness, various paræsthesiæ, formication, itching, or more or less diminution of sensation. Very rarely true anæsthesia is found. The peripheral nerves are not specially involved save, as already stated, in the form of neuralgia or neuritic pains. Very rarely, however, a local palsy is observed. Thus Westphal, also Paulowski, noted facial paralysis. Inequality of the pupils, which has been already mentioned, was noted by Hertzog.

A close study of well-marked cases of diffuse scleroderma soon convinces the observer that the skin is not the only structure affected. The loss of the superficial fat enables one in many cases to feel the subdermal structures with considerable accuracy. It is frequently found that these are more resistant to the feel than they should be. As has been already stated, the impairment of motion in certain joints frequently bears no definite relation to the degree of the involvement of the superjacent skin. The conviction forces itself upon one that the same process which is instrumental in bringing about the induration and contracture in the skin is also instrumental in bringing about induration in other structures, as, for example, in the connective tissue about the joints, the deep fascia, the tendons, and the muscles. This involvement of tendons and muscles has been observed by Ball, Thibierge, Kühler, and by the writer, and has been demonstrated pathologically by Foullerton. Muscular atrophy, apparently from disuse, has also been occasionally observed. Sometimes special groups of muscles are involved; thus Thibierge found the biceps, supinator longus, and sternomastoid converted into hard cords. Similar changes have been noted by Lewin in the sternomastoid and by Leflaive in the pectoralis major. Contractures of tendons have also been observed by Hall and by Sympson.

Instead of being evenly diffused over various portions of the face, neck, limbs, and trunk the sclerotic process may, as already stated, be accentuated in, or limited to certain areas. Indeed, diffuse universal scleroderma is relatively infrequent and occurs, according to Lewin and Heller, in about fifteen per cent. of the cases. Regarding the frequency with which different portions of the body are affected with scleroderma, it would appear that the upper extremities suffer most frequently of all, next the trunk, then the face and head, while the lower extremities suffer least. Very rarely certain peculiarities of location are noted; thus in two cases the sclerodermatous process was limited to one-half of the body. Again, in a few cases the affection was found localized upon the flexor surfaces of the limbs, in others

upon the extensor surfaces. It may here be noted that the palmar and plantar surfaces are affected with the greatest rarity. Such induration has, however, been noted by N. Moore and also by the writer.

Very rarely the sclerotic process involves the mucous membrane of the mouth, of the tongue, of the gums, and even of the vagina, though as regards the latter it should be stated that scleroderma of the genitals either in the male or female is excessively rare. As regards the more limited forms of scleroderma, that is those in which the process occurs in strips or patches of variable shapes and dimensions, it is necessary to state that they may or may not follow certain nerve trunks in their distribution. The number of cases in which such a relation to either a sensory, a motor, or a mixed nerve is apparent, is outweighed by an equal or even larger number of cases in which no such relation can be discerned. That, however, in certain cases there is a very close relation between the sclerodermatous process and certain nerve trunks cannot be denied. Thus the process is noted now and then in close relation with the various branches of the trigeminus. At other times it has been noted to follow closely the distributions of one or more intercostal nerves. In one case it followed closely the course and distribution of the sciatic. To multiply these instances is unnecessary. They are sufficient to indicate the very important, though apparently erratic, rôle played by the nervous system. As already stated, the cases of limited scleroderma, in which no relation can be demonstrated with nerve trunks, fully outweigh those in which such a relationship is clear and undeniable, and if the nervous system plays a rôle in the production of these forms, the seat of the lesion must be referred to the nervous centres rather than to the nerve trunks.

The circumscribed forms of scleroderma present features similar to those of the diffuse form, save that the skin changes are limited to certain areas. General symptoms are much less pronounced, although tingling, neuralgic pains, hyperæsthesia, or other signs indicative of nervous involvement may be present. The patches vary greatly in size and in shape. Thus they may be so small as to be readily covered by the tip of the finger or so large that they can with difficulty be covered by the hand. The patch may be irregularly elongated, rounded, hyperæmic, purplish, or violaceous in color. All shades of pigmentation may be present. At other times no trace of pigmentation can be seen, the surface being absolutely white. The patch may sometimes have a waxy, at other times an ivory appearance. Sometimes it is smooth and shining, at other times dull in appearance. Not infrequently it is discolored by yellowish patches of dead

epidermis. At an early stage it may be slightly elevated, but later during the atrophic period it may be either on a level with the surrounding skin or slightly depressed. It may become excessively thin and contracted or may present the appearance of parchment. In some cases the patches present the appearance of elongated depressed cicatrices.

Morbid Anatomy.

When we come to analyze the post-mortem findings, we find, first, as to distribution that the changes are by no means limited to the skin and subcutaneous tissue. As we have already seen, changes in the joints and in the tissues about them can be inferred from many of the clinical phenomena, and is well shown in Roentgen pictures. That changes in the joints occur, particularly about the hands, has been demonstrated more especially by Legrange, who observed in sclerodactyle union of apposed joint surfaces by fibrous tissues, loss of articular cartilage and calcareous deposits in the fibrous tissues about the joints. Similar joint changes were also noted in the case studied by Verneuil and Mirault, in which short fibrous bands extended between the apposed joint surfaces, and in which there had been destruction of synovial membrane. Changes in the bones, at least of the fingers, are also at times to be inferred from the clinical findings. Actual examination of the tissues has revealed in the hands of Wolters an interstitial inflammation of the bone, while Lagrange describes a disseminated inflammation affecting periosteum, bones, and joints. Changes have also been noted in the muscles and other tissues. Foullerton, who examined the amputated leg of a case of scleroderma, infers from the widespread character of the change that scleroderma is primarily a disease of genetically related groups of tissues of mesoblastic origin; that is, of the derma, the subcutaneous tissue, the muscle fibres, and the fibrous capsules of the joints. Varied changes have infrequently been noted in tissues other than those here mentioned. They appear to be without special significance, and have been noted in the lungs, the heart, the liver, the peritoneum, the kidneys, the spleen, the intestine, and the aorta. Cardiac changes seem to have been noted rather more frequently than others. Thus out of twenty-eight cases collected by Lewin and Heller some change, such as hypertrophy, degeneration, myocarditis, or pericarditis were noted in fourteen cases.

The result of the microscopical examination of the skin in scleroderma is well known, and consists of an increase of the connective-tissue elements, together with sclerotic or inflammatory changes in the blood-vessels. That similar changes take place in other tissues

is not only probable, but evident from the examinations which have thus far been made. It is, however, extremely doubtful whether this affection can be considered primarily as a sclerosis. Certainly the phenomena presented by cases in which we have a recurring, or even a shifting infiltration, and which sometimes pits or yields upon persistent pressure, shows that we have every reason to believe that the changes found in the tissues microscopically are terminal changes. The clinical phenomena certainly justify the inference that primarily the connective tissue is infiltrated, swollen, or enlarged. The single fact that the infiltration may vary considerably from time to time, that at times the skin is soft and flexible, and at others densely infiltrated, shows that some variable or changing process not consistent with that of a primary sclerosis is at work. That a change does take place in the connective tissue other than a primary sclerosis is also held by Lewin and Heller, who regard it as very conceivable that the enlargement of the individual fibres of connective tissue depends upon their infiltration with an albuminoid substance—a saturation with some coagulable material. Unna holds that the changes are primarily in the connective tissue and especially in its intercellular or ground substance.

A view that presents itself as plausible is that the primary change in scleroderma is an increase or chemical modification of the collagen of the skin and subjacent tissues. Certainly the initial change appears to be a swelling and infiltration of the collagenic tissues. We have here a certain analogy between this disease and myxœdema. That myxœdema is accompanied by connective-tissue changes, and that there is also an infiltration of the connective tissue with mucin, a substance normally present in the intercellular or ground substance, is well known, and it would seem that just as in myxœdema there is an increase of the intercellular mucin, so we have in scleroderma an increase of the collagen or some as yet unknown chemical modification of this substance, which increases the bulk of the tissues containing it. That, however, these changes differ radically from the changes in myxœdema is shown not only by a microscopic examination of the skin, by the symptomatology and by the clinical history, but also in the difference following the administration of the thyroid extract. In four cases collected by Lewin and Heller no improvement followed the administration of this remedy, so powerful in myxœdema.

Whether or not the change in the connective-tissue elements is dependent upon the faulty action of some ductless gland, as is the case in myxœdema, must, for the present at least, remain a matter for speculation.

The theory that the disease is a vasomotor neurosis explains nothing. It merely states in a vague way that there is some relation between the changes in the skin and the condition of the central nervous system. That, however, some special as yet unknown condition exists independent of the nervous system is proven by the various facts of etiology. Thus a severe physical shock to the nervous system appears to be a frequent exciting cause. Evidently, however, such a cause cannot be the real factor at work, for in such case scleroderma, instead of being a rare affection, would be an exceedingly common one. It would be as common, indeed, as traumatic neurasthenia. The same is true of psychic shock. Certainly scleroderma must be looked upon as among the rarest of the sequelæ of psychic shock, and so it is with the cases which follow exposure to cold. If cold were a potential factor in the production of scleroderma, how common an affection it would be! Every winter, every climate, every avocation in which exposure to cold was attendant, would bring with it a history of scleroderma. Such a relation, it is needless to say, does not exist. Other causes which are every now and then assigned, such as muscular overexertion, severe labor, menstrual disturbances, pre-existing infectious diseases, need only to be mentioned to show how rare they must be. The findings as regards the urine and the blood have already been dwelt upon and do not appear to be especially significant. As already stated, in one case studied by the writer in the œdematous stage an excess of waste products was being eliminated, while in another studied in the atrophic stage the waste products were enormously diminished. Especially was this true of the urea. The increase in the leucocytes occasionally observed in the blood has a general significance only, as has the presence of albumoses.

Diagnosis.

The diagnosis of scleroderma rarely presents any difficulties. In the diffuse form attended with œdema, a superficial resemblance to myxœdema is perhaps suggested. However, the marked difference in the character of the infiltration, which in scleroderma is resistant and in myxœdema is soft and yielding, would enable us to distinguish between the two affections. Further, the countenance, in spite of the swelling, is entirely different in the two affections. In myxœdema the patient presents a sleepy, puffy look, while the face has the appearance of a full moon. In scleroderma the face has a mask-like expression in which there is no trace of somnolence. In myxœdema also the hands become broadened and the fingers thickened, sausage-shaped, or clubbed. In scleroderma the hands either remain

unaffected or present sooner or later the condition above described as sclerodactyle which is so characteristic as not to permit of error. In myxœdema also, the hair becomes thin and brittle. This is not the case or very rarely the case in scleroderma. In the atrophic period of diffuse scleroderma, as we have seen, the countenance becomes so changed as to be pathognomonic, and confusion with myxœdema or other affections is impossible. The circumscribed forms of scleroderma also present little or no difficulty, and for the facts necessary to diagnosis the reader is referred to the section on symptomatology. There is but one affection with which it could be confounded and this is progressive facial hemiatrophy. The symptoms presented by the latter disease so closely resemble those of scleroderma circumscripta that many physicians regard them as being the same disease. The disease generally begins in the distribution of the trifacial nerve. As a rule a whitish patch is noted over the cheek or over the lower jaw or near the zygomatic process. The patch feels hard and seems to have lost its elasticity. Gradually it becomes firmer, more dense, and begins to sink below the general level of the surrounding skin. Soon a pit or depression is formed which is quite marked. Gradually the subjacent tissues, muscles, and bones become atrophied, apparently because of the pressure of the hardened and contracting skin above. The reader will observe that the differentiation between scleroderma and hemifacial atrophy is not by any means clear and that after all hemiatrophy may be a circumscribed form of scleroderma. From what, however, is known of hemifacial atrophy it appears to depend upon some perverted or defective action of trophic fibres in the trigeminal nerve, and there would appear to be a closer relation to nerve distribution in this instance than is observed ordinarily in scleroderma.

Prognosis.

As already stated, scleroderma either terminates in recovery or improvement, or it progressively increases in severity until death. The relative frequency of these results has also been stated above (p. 530). It is impossible in given cases to predict which of these results will ensue. Gradual softening and resolution, together with improvement in the general bodily condition, are to be regarded as of good omen. Rapid disappearance of induration is to be regarded perhaps with apprehension, inasmuch as we are aware that it may lead to excessive atrophy of skin and perhaps to destruction of subjacent tissues. Data for accurate prognosis are not present in any case. The prognosis of sclerodactyle is almost always unfavorable.

It should, however, be borne in mind that cases in a very early stage may undergo spontaneous recovery, but that if the parts have been more or less fixed for a long time recovery is practically impossible. Sclerodactyle must be regarded as largely a terminal condition.

Treatment.

Every plan of treatment presupposes some special knowledge of the pathology of the affection with which we have to deal. In scleroderma, however, as we have seen, definite pathological data are still wanting and we are forced to rely upon general measures. Occasionally definite good seems to ensue upon the proper application of the latter. Thus massage and passive movements seem at times to assist in the resolution of the infiltration and in preventing the fixation of parts. That they do good in many cases there can be no doubt. In others, however, especially if the measures be applied too vigorously, they seem to do harm. Hydrotherapy has also been employed but with distinctly less benefit than massage. General tonic remedies are also indicated. The diet and digestive tract also should be carefully watched. As a rule patients suffering from scleroderma, especially of the diffuse form, do better upon a diet which is plain and unstimulating and to which milk has largely been added. According to the case, also, various forms of exercise should be instituted and the restricted motions of the various joints restored as much as possible. We should also take the hint afforded by the quantitative examination of the urine, namely, that the output of waste products is defective, and should give liquids in relatively large amounts so as to promote their excretion by the kidneys. Thyroid extract which is so potent in myxœdema, appears to be of little value in scleroderma. In some cases, however, it has been given with apparent benefit, especially in the early or œdematous stage, but it is probable that this apparent benefit is the result not so much of the action of the thyroidin upon the infiltration as of the atrophy of the subcutaneous fat. In four cases collected by Lewin and Heller no benefit followed the use of the thyroidin. We should remember, also, that the remedy is capable of great harm—that by reason of its destructive action on fatty tissue it may hasten and favor to a dangerous degree the progressive emaciation of the patient. It is furthermore to be borne in mind that thyroid extract is a powerful febrifacient, especially if used in large doses. If we give thyroidin at all we should give it in small doses and should carefully study the temperature of the patient, and at the first indication of fever the drug should be withdrawn.

The treatment of the special symptoms must be based upon general principles. The neuralgic pains should be controlled so far as

possible by the coal-tar derivatives; morphine should only exceptionally be used. The joint pains when present appear to be favorably influenced by the salicylates and by piperazin. Should painful ulcers make their appearance they should be treated by sedative applications and, if necessary, by the local use of cocaine. Large ulcers are, as we have already indicated, very infrequent. Much more common are slight ulcers about the joints of the fingers in cases of sclerodactyle. These lesions which are evidently trophic in character, heal with great difficulty, even under antiseptic precautions. The writer has, however, had considerable success in the treatment of the ulcers by saturating their surfaces with bovinine or by applying pledgets of cotton wet with this preparation. After a longer or shorter period granulations appear and soon reach the general level of the skin, when an epithelial covering is formed and nothing but a small white patch finally remains to indicate the former presence of the ulcer.

All measures which tend to increase the body weight also favor improvement, and in cases in which the disease appears to have come to a standstill a modified form of rest treatment may be instituted. As stated in the section on symptomatology, the weight of the patient is as a rule far below normal. It is noticeable that an increase of weight is followed by a marked improvement in the skin and other surface structures. This is shown especially by a more luxuriant growth of the hair and in a more rapid growth and healthier appearance of the nails.

In many cases no matter what measures we institute, the disease steadily progresses, joint after joint may become fixed and distorted until finally the patient is utterly unable to attend to his personal wants. These results may follow even if massage and passive or active movements have been faithfully persisted in.

ACROMEGALY.

Acromegaly is a disease which, as the name implies, presents as its principal symptom an enlargement of the ends or extremities of the body. It was first described by P. Marie in 1886 and it is consequently also known as Marie's disease. Von Recklinghausen has proposed the term pachyachrie—thickness of the ends of the body—but this has not come into general use. Acromegaly is a chronic affection, in which especially the hands, the feet, the face, and the head become greatly enlarged. The enlargement is not, however, limited to the extremities, but involves to various degrees other portions of the body as well.

Since Marie's original description appeared, numerous cases have been reported and various articles have been written on the subject. The most important, however, are an exhaustive treatise upon the affection by Souza-Leite in 1890, an exceedingly valuable article by Dr. Joseph Collins of New York in *The Journal of Nervous and Mental Disease*, December, 1892, and January, 1893, and an able summary by Osborne in the "Reference Handbook of the Medical Sciences," Vol. IX., 1893.

Etiology.

Nothing special is known regarding the etiology of acromegaly. In the first place it may be stated that heredity plays no rôle whatever, nor can any other element be traced in the family history. For instance, the writer has studied cases of well-marked acromegaly occurring in families in which the other members were absolutely normal and in which no nervous or mental disease can be traced. It is not uncommon for the brothers or sisters of an acromegalic patient to be small-limbed and small-boned, so that the patient's appearance is strikingly different from that of the rest of the family. As regards sex it is evident from the facts at our command that the disease occurs equally among males and females. It may also occur in any race, no race being exempt. It usually begins in early adult life, the majority of cases being discovered between twenty and forty years of age. Sometimes, however, it is noted in early adolescence or shortly after puberty. It is said, indeed, even to occur in infancy. Old age, on the other hand, is not exempt. It has been known to develop after sixty years of age. No congenital factors appear to play a part in the evolution of acromegaly, nor do any of the diseases of childhood or adolescence. Indeed it may be stated that no facts of importance relative to other diseases can be elicited from the personal history. Thus infectious diseases, trauma, shock, and emotional disturbances play no part in the evolution of this strange disease. It is true that occasionally such an apocryphal cause as rheumatism or malaria is assigned by the patient, but in reality these affections bear no relation to acromegaly. In like manner as in other affections of obscure origin, such vague causes as overwork and exposure, hysteria, and alcoholism have been recorded as playing a rôle. As might be expected it occasionally occurs along with other affections, but these are so various that no connection between them and acromegaly can be made out. Thus, it has been seen occurring with diseases of the spinal cord, such as locomotor ataxia and syringomyelia, and also with the various psychoses. Sometimes it has been noted along with other diseases with which a possible relationship is at first sight sug-

gested, such as elephantiasis, gigantism, and arthropathies, but no definite relation can be shown to exist. In short no conclusions can be drawn in the present state of our knowledge relative to the mode of origin or primary causation of acromegaly.

Symptoms.

It is very difficult as a rule to fix the time of onset of the symptoms. The disease begins so gradually that frequently several years

Fig. 57.—Acromegaly (from a photograph).

pass by without the affection being recognized. The friends of the patient, if the increase in size of the face and hands attracts their attention, are apt to imagine that he is of unusual physical development, and, as was the case in one of the patients of the writer, that he must be very strong. It frequently happens that the disease is not discovered until some incidental symptom or intercurrent malady

leads the patient to consult a physician. At other times the affection is discovered merely by accident. Thus two cases described by the writer were discovered by him on the street and had not presented any symptoms suggesting medical care. While the appearance of the acromegalic subject is often such as to suggest to the superficial observer great strength (see Fig. 57) the first symptom noted is frequently the great disparity between the size of the limbs and the amount of strength revealed. We learn also that subjectively the patient experiences a sense of weakness which is at times quite profound, and this frequently leads to a general indisposition to exertion. In a few recorded cases, however, it should be stated that strength appears to have been somewhat increased.

In addition to the striking enlargement of the face and hands, which are among the earliest facts to attract our attention, it is noted that the attitude of the patient, in the well-developed disease, is quite peculiar. He stands with the head thrown forwards and with the chin slightly tilted upwards, while the back is "humped" by a more or less marked cervicodorsal kyphosis. The shoulders are rounded, while the arms, owing to the spinal curvature, are apparently increased in length and often add strikingly to the clinical picture. Owing to the spinal curvature also, the stature becomes lessened—sometimes to a marked degree.

On seeing a case of acromegaly, as just stated, we are at once impressed by the massiveness of the features and by the enormous size of the hands and feet. We observe that the nose is very large and that the chin projects to an unusual degree, that the cheek bones are exceedingly high, that the supraorbital ridges are unusually prominent, while the face as a whole has very unusual length and breadth. In like manner we are impressed with the great breadth and unusual thickness of the hands and fingers, and when we turn our attention to the feet we find that they likewise are of excessive size. When we question the patient he tells us that he has noted for a long time past that his head, hands, and feet were growing larger. He may say that he has constantly been required to buy larger hats, larger gloves, and larger shoes, and yet this change is so gradual that the patient is as a rule unable to fix a time, even approximately, when the disease began to make its appearance. Valuable information can, however, occasionally be obtained on this point from old photographs, and frequently it is possible by this means to fix the ages between which the enlargement first made its appearance. When the patient seeks medical advice it is most frequently because of headache, which in a large number of cases is a prominent symptom. It is apt to be more or less constant and of increasing severity and suggests sooner

or later headache of organic origin. It is most frequently frontal, though with time it generally becomes widely diffused. At times instead of headache or occurring with it the patient suffers from pains in the joints, or from curious paræsthesiæ, or from a sense of weight and discomfort in the legs and arms. Not infrequently among the early symptoms noted are disturbances of vision and curious defects of the visual field. Any one of these symptoms may cause the patient to consult a physician and thus lead to the discovery of the disease.

When we inquire into the previous history of a case we are likely to find it negative save possibly as to the progressive increase in the size of hands and feet. In women, however, the disappearance of the menses is noted and this is as a rule an early symptom. In men diminution or loss of sexual power may be observed. Rarely we obtain a history of profuse and recurring epistaxis. More frequently a history of somnolence is present and this may occur as a very pronounced symptom, the somnolence extending over days and often weeks.

Among interesting symptoms observed in the larger number of cases of acromegaly are increased thirst and increased appetite. Sometimes the amount of food consumed is very great, while thirst is increased to such a degree as to suggest the thirst of diabetes. This symptom often attracts the attention of the patient more forcibly than the increase of appetite and he may mention the fact voluntarily. In keeping with this increased thirst the amount of urine voided is greatly increased, as is also the perspiration. This is apt to be excessive, especially about the head and neck. In a very small number of cases sugar has been found in the urine and in a still smaller number albumin. These facts are, however, without special significance.

When we examine the patient in detail we note that the nose is massive, though as a rule it is not distorted. The increase in size appears to affect all of its tissues, the nasal bones, the nasal cartilages, and the soft parts; all are much thickened and the anterior nares are wide and unusually capacious. The size of the lower jaw is perhaps the second most striking feature. The body of the bone appears to have grown greatly; it has evidently become broader and thicker, especially the alveolar portion and the chin. The enlargement generally involves the rami less than the other portions, although occasionally enlargement of the rami is also noted.

As might be inferred, the teeth of the lower jaw become displaced, especially the incisors, which are carried forward so that in closing the jaw the lower incisors are no longer overlapped by the upper in-

cisors but project beyond them. The space between the teeth is often increased so that in advanced cases they seem to stud the gum at intervals. Sometimes loss of the teeth ensues. The teeth are not involved in the enlargement, though in a case described by Cenas in which the disease appears to have been congenital, the teeth also were increased in size. The supraorbital ridges are, as already stated, much thickened and very prominent, and this causes the forehead to appear low and retreating. The malar bones are exceedingly prominent while the cheeks themselves are flattened and drawn. The lips are invariably enlarged, especially the lower lip which is always hypertrophied in excess of its fellow. It is much thickened and sometimes everted. The nasolabial folds are often accentuated, giving an expression of grief to the patient. The skin of the forehead and face is thickened and coarse and is often darkened. Other structures, such as the eyelids and ears, also usually partake in the general increase in size. This is less noticeable as a rule of the eyelids than of the ears, but the latter are sometimes strikingly enlarged and this enlargement appears to be due not only to the hypertrophy of the skin but also to the cartilaginous structures. The upper jaw and the other bones of the face do not as a rule take part in the enlargement —at least they do so to a relatively small extent. In many cases, it cannot be denied, the superior maxillary bone is enlarged, but this enlargement is never in proportion to that of the lower jaw, the nose, or the cheek bones. Sometimes the eyeballs are unusually prominent as though they themselves were enlarged or as though the cavity of the orbit had been diminished by overgrowth of bone. In other cases, again, the eyes appear less prominent than normal. At times they appear even retreating and deeply set, apparently because of the increase in size of the supraorbital ridges, the malar bones, the zygoma, and the nose.

When we ask the patient to protrude his tongue we find that the latter is also hypertrophied, sometimes enormously. It is exceedingly broad and thick and this increase in size may give rise to a certain awkwardness and clumsiness of speech, the latter becoming somewhat slow and indistinct, especially as regards syllables in the formation of which the tongue is especially concerned. The tongue is rarely smooth, more frequently it is deeply furrowed. As a rule it presents the indentation of the teeth. Hypertrophy of the half arches and uvula may also be found. Even the epiglottis and the larynx may be involved and in such cases the quality of the voice is entirely changed, becoming coarse and of lower pitch. The neck may be found thick and short, but more frequently it is disproportionately small. As already stated, it is bent more or less forwards

and takes part in the anterior curvature of the spine which also involves the dorsal region.

When we turn our attention to the hands we note in the first place that they are excessively broad and much thickened. Often they appear somewhat lengthened. In curious contrast with the enlarged hands we frequently see, at least in the earlier periods of the disease, the forearms comparatively small or even normal in size, though later on these also may become massive (see Fig. 57). As a rule, however, the forearm is relatively small and the enlargement of the hands is therefore the more striking. The joints of the hands are prominent, the skin, subjacent connective tissue, and fat are excessively thickened. On the palmar surface, the transverse lines are seen to be much accentuated. The ulnar border of the hand is invariably somewhat more hypertrophied than the other portions. The fingers are diffusely and evenly enlarged and have for this reason been termed sausage-shaped. The nails appear to partake in the enlargement to a less degree than the other structures. Consequently they appear relatively small. They are flat, longitudinally ridged or striated, and frequently are brittle. The growth of the hair on the dorsal aspect of the hand and fingers does not appear to be especially interfered with. This is true also of the hair of the head and face. When we turn our attention to the feet, we find that they present features similar to those observed in the hands. Not only is the foot broader but the toes are thickened, especially the great toe, while the heel projects backwards to an excessive degree, giving a negroid appearance to the foot. The arch of the foot also is lost, the patient becoming more or less flat-footed. The malleoli are much hypertrophied, and the tendo Achillis is thickened and unusually prominent. The outer edge of the foot, like the ulnar edge of the hand, is especially hypertrophied. The great toe also is more hypertrophied than are its fellows. The skin, like the skin of the hands, is hypertrophied and thickened. The nails are affected similarly to those of the fingers.

If we examine in detail the trunk, we find as the most striking anomaly presented, the cervicodorsal kyphosis already mentioned. In addition to this kyphosis some scoliosis, rarely marked, may be noted. In examining the spinous processes it is frequently noted that their ends are unusually thickened and prominent.

The shoulder girdle also presents signs of involvement, the clavicle is frequently much enlarged and thickened at its sternal end. The examination may also disclose that the sternum is hypertrophied, that it is much broader and more prominent than normal. Hypertrophy may also be especially evident on examination of the xyphoid

process. In shape the sternum is sometimes found distorted and irregular, owing doubtless to irregularity in the hypertrophic process. As would be expected, the ribs may also partake in the hypertrophy. They are wide, evidently enlarged, while their costal cartilages feel hard and inelastic. Sometimes their surface is irregular, numerous nodosities being present. The chest as a whole is increased in size, especially in the anteroposterior direction. Contrary to what we would expect, the abdomen is not full and enlarged, but if anything somewhat flattened. The hips may also present more or less marked enlargement. The pelvis as a whole seems broader and the hip bones are prominent. The pubis also is especially prominent, abnormally high, and hypertrophied.

Passing now to the arms we find that there is little or no change noticeable in the shoulders, although in a few cases these have been reported enlarged. The upper arm is small or merely normal in size. The same statements are true, as a rule, regarding the forearm. However, every now and then the elbow is much enlarged as is also the lower third of the forearm. In other cases again, as has already been said, the entire forearm is hypertrophied, the hypertrophy being most marked on the ulnar aspect. These changes are, however, rather the exception than the rule. The thigh rarely, if ever, shows enlargement save in the region of the condyles of the femur. The same is true also of the leg; although here the malleoli partake of the general hypertrophy of the foot. The patella at times is much enlarged.

Marked hypertrophy of the penis, of the labia majora, of the labia minora, or of the clitoris may exist and in one of the cases of the writer the testicles were enormously enlarged, attaining the size of these glands in the sheep. The vagina also may be lengthened.

The station and posture of the patient have already been sufficiently dwelt upon. The station betrays no increase of sway. The gait is often lumbering and heavy. The movements of the arms and especially of the hands are clumsy and awkward. When we test the patient's strength we find that he is relatively weak, the enlarged hand is incapable of giving the powerful grip which its size would suggest. This discrepancy becomes at once apparent and is in consonance with the patient's statements of fatigue and incapacity for exertion. The muscles of a part, for instance of the forearms, may be, as we have seen, much enlarged and yet they are soft and compressible. Tremor or fascicular twitching is, however, not noted. The knee-jerks and other tendon reactions present no noticeable change. The electrical reactions of the muscles are normal as are also those of the nerves.

When we examine the patient for sensory phenomena we find that none are presented by the skin. Everywhere sensation is normal, but while true anæsthesia is not present, small objects appear not to be as readily recognized by the patient as in health. When we examine the special senses, interesting changes may be discovered. Especially is this true of vision. Diminution of the visual fields, a diminution which takes various forms, sometimes that of a bitemporal hemianopsia, is not infrequently present. At times, especially in advanced cases, absolute blindness is noted. In other cases again the eyes appear to be relatively normal. The pupils as a rule present no peculiarities, although infrequently they are found to be dilated and sometimes slow in their reaction to light. Nystagmus also has been noted in a few cases. According to Marie, the optic nerve may at an early stage present the signs of a neuritis. On the other hand, optic atrophy, more or less marked, has not infrequently been found. As a rule, the sense of hearing presents no anomalies whatever. Rarely, however, deafness is noted, and at other times tinnitus aurium. Anomalies of smell and taste are rarely if ever observed.

Among the subjective phenomena the symptom most frequently met with is headache, which, as already stated, is of such a character as to suggest organic intracranial disease. In other cases, again, it is either entirely absent or of so slight a character as to be revealed only in response to questions. At other times the pain is of great severity. When present the headache is referred to the forehead or to the top of the head, though as the case progresses it becomes widely diffused. In addition to the headache acromegalic patients often complain of pain in the larger joints, especially the knee joints. Sometimes, though less frequently, pains are referred to the fingers or feet. Pains, shooting in character, may also be referred to various portions of the trunk. Numbness or pins-and-needle sensations are sometimes noted, though they are rarely marked.

Somnolence, which has already been mentioned, is also a not infrequent symptom. The somnolence appears to come on in attacks which may last for weeks at a time, the symptom being entirely absent in the intervals. In other cases, again, it is present in a slightly marked degree all of the time. The patient frequently states also that he suffers from giddiness, though this rarely amounts to actual vertigo. As a rule no other symptoms of involvement of the nervous system are present. Occasionally, however, the patient is depressed, hypochondriacal, or melancholic, or he is markedly apathetic, heavy, and indifferent. Not infrequently it should be stated there is a distinct loss of memory. Dementia, however, is never noted; in fact it

should be clearly impressed upon the reader that in the larger number of cases no mental symptoms of any kind are observed.

Pathology and Morbid Anatomy.

As might be expected, autopsies upon cases of acromegaly reveal more or less marked hypertrophy of the bones of the hands, of the feet, and of the face—especially of the lower jaw. Other bones are found enlarged to various degrees. The most interesting findings, however, consist in hypertrophies of various glandular structures, more especially of the pituitary body. This structure is so frequently found enlarged that it is difficult to resist the conviction that it is in some way related to the disease. Marie suggested that the pituitary body bears a relation to acromegaly similar to that which the thyroid gland bears to myxœdema. It should be borne in mind, however, in this connection that in a very small number of cases the pituitary body has presented apparently no changes (notably a case of Virchow). Numerous other glands occasionally participate in the enlargement, more especially the thyroid and the thymus glands, while hypertrophy of the lymphatic glands, the spleen, the kidneys, the lobes of the liver, and even of the testicles is not uncommon. However, as already stated, the enlargement of glands other than the pituitary is so inconstant and relatively so infrequent that these changes must be considered as of secondary importance. Dulness on percussion over the sternum during life was thought, especially by Erb, to indicate enlargement of the subjacent thymus gland, but the dulness appears to be due really to the hypertrophied and thickened bone. The changes found in the nervous system are inconstant and without significance. Marie, for instance, detected hypertrophy of the sympathetic system and peri- and endoneuritis. No changes of importance are found in either spinal cord or brain. The skin, as already stated, is hypertrophied, especially over the affected portions of the body. The subcutaneous connective tissue may also be increased, but at a later period it—the subcutaneous fat at least—becomes much diminished. The muscular tissue itself is as a rule found atrophied, though occasionally, as would be inferred from the clinical facts, hypertrophy of this tissue is revealed.

As regards the bones it is noted that the enlargement affects principally the distal ends of the long bones of the limbs and the bones of the carpus, metacarpus, and fingers, and of the tarsus, metatarsus, and toes. It is the spongy tissue which is principally affected, although the tissue of the shaft of the long bones may also be increased. Occasionally, in addition to the hypertrophy of bone, bony deposits

may be found in the joints and in the cartilages. The fact that increase in the length of the bone does not take place save in cases associated with gigantism, depends probably upon the period at which the disease begins, namely, after the epiphysis and the shaft have become more or less united. The examination of the bones in detail reveals facts closely in keeping with the clinical observations. It should be borne in mind that no bone is free from possible involvement.

Examination of the blood reveals no abnormalities.

The facts at hand are as yet insufficient to warrant a satisfactory hypothesis regarding the nature of the disease. We can pass over without comment the theory of Freund, that the disease is one of development and consists of a return to a primitive type. The theory of Klebs that the thymus gland plays a special rôle is also based upon insufficient evidence, inasmuch as the number of cases in which thymus enlargement is present is relatively small. The theory of von Recklinghausen, that it is due to a vasomotor neurosis, is somewhat more acceptable, and yet, like all general explanations, is exceedingly unsatisfactory. While it is perfectly true that many of the symptoms, such as excessive perspiration, polyuria, etc., lend support to this theory, no explanation of the origin of the neurosis is given, nor are we told why this special form of dystrophy should result rather than the various other trophic disturbances with which we are clinically also familiar. At present we are constrained to accept the theory which was first suggested by Marie, namely, that the pituitary gland plays an essential rôle in this disease. Andriezen has thrown some light upon the probable function of the pituitary body by studies in general morphology. He has shown, for instance, that the subneural gland in the larval amphioxus is the analogue of the pituitary body in the higher animals and in man. His results are thus summarized by Collins ("Nervous Diseases by American Authors," p. 893): "He believes that the *ensemble* of evidence proves that the pituitary gland is not a simple structure having one simple function, but a complex organ composed of three parts: (*a*) An anterior secreting glandular organ; (*b*) a water-vascular tube lined with ciliated epithelium and connecting the buccal cavity with the ventricles and the rest of the neural cavities; and (*c*) a posterior sensitive nervous lobe. The last two are well developed and functionate in ancestral vertebrata, but become obliterated and atrophied in structure and function in all forms above larval acraniates and ammocetes. In man the posterior lobe represents little beyond a neuroglia remnant of what was once a functional portion of nerve tissue in ancestral vertebrata. The glandular secreting portion (anterior lobe) is the type of

a secreting structure of epithelial cells arranged in lobules and acini with many ducts opening into one principal duct. Its secretion is carried with the water-vascular stream through the central nervous system, and the action of that secretion must be either a trophic one on the nervous tissues or it must have a destructive effect to neutralize waste products resulting from the activity of nerve tissues."

As showing the findings in acromegaly, the interesting examination of a case by Mosse and Daunic (*Bulletin de la Société Anatomique de Paris*, 95, LXX., p. 633) may be cited as typical. These observers found enlargement of the pituitary body and of the thyroid and thymus glands. The enlargement of the pituitary body appeared to be sarcomatous in character. The thyroid body presented several changes: exaggeration of the colloid substance, cystic formations, and diminution of vascular proliferation of the fibrous tissue. The thymus gland, on the other hand, presented simply hypertrophic enlargement.

Diagnosis.

Acromegaly can as a rule be readily recognized. The enormous exaggeration of the features and the great size of the hands and feet in well-developed cases leave little room for error. However, it not infrequently happens that cases in which the disease is not far advanced present themselves. The unusual size of the features, the unusual development of the special bony processes, such as the supraciliary ridges and the inion, while the skull itself remains unchanged, should excite suspicion. Similarly if the hands are much larger than normal and if the fingers, instead of presenting the taper of normal fingers, are somewhat sausage-shaped, if the ulnar edge of the hand is thickened out of proportion, especially if the hand is excessive in size as compared with the forearm, the diagnosis of acromegaly is practically certain. Changes in the face and in the hands of course suggest an examination of the feet. If the feet are excessive in size in proportion to the leg; if the instep is thickened; if the toes are hypertrophied, especially the great toe; if the outer edge of the foot is disproportionately enlarged, and especially if the heel is unusually prominent, the diagnosis must be looked upon as established. Especially will this be the case if, in addition to the peculiarities of the face and extremities, the patient presents the history of having been obliged for several years past to buy hats, gloves, and shoes of ever-increasing size. Further, if the patient gives an account of severe headache, of headache that is continual, that presents little or no variation; if he is at times somnolent and suffers from vertigo; if he presents anomalies of the visual fields, has excessive thirst or excessive hunger, no room is left for doubt.

Acromegaly must be differentiated from certain other affections, *e.g.*, gigantism. In gigantism there is as a rule more or less symmetrical and proportional overgrowth of all portions of the body. The features are not excessive as compared with the head and neck. The hands are not enlarged out of proportion to the arms, nor the feet to the legs, but on the contrary, the hands and feet are disproportionately small. In acromegaly the overgrowth occurs at the ends of the extremities, at the distal ends of the long bones, and in the ends of the various bony processes. It must be remembered, however, in differentiating between these affections that now and then, though rarely, gigantism and acromegaly occur together.

From myxœdema acromegaly is readily differentiated. In myxœdema there is an evenly diffused swelling in the subcutaneous tissue all over the body. This swelling is soft and does not pit on pressure. The features are not enlarged but the entire face becomes rounded and "moon-shaped." The hands and fingers are swollen; the latter are club-shaped. There is no disproportionate enlargement of the ulnar edge of the hand. Myxœdema occurs most frequently in women, eight out of ten cases being female; in acromegaly the sexes are equally affected. Again, myxœdema begins at a distinctly later period of life, generally at forty or fifty years of age, while acromegaly begins most frequently between the ages of twenty and forty. In myxœdema the bones are never enlarged; in acromegaly they are always enlarged. The skin in myxœdema is soft and not discolored; in acromegaly it is coarse, hypertrophied, hairy, and sometimes pigmented. The possible though rare concurrence of the two diseases should be borne in mind. One such case has been reported by S. Solis-Cohen.

Leontiasis ossea is a disease which should also be mentioned in discussing the diagnosis of acromegaly. In leontiasis ossea there is no exaggeration of the features such as is seen in acromegaly. There is no hypertrophy of the supraciliary ridges or inion, but there is on the other hand, an enormous and diffuse hypertrophy of the skull or there is an irregular heaping up of bony deposits. The extremities are not affected at all.

Acromegaly can be readily distinguished from the trophic disorder described by Marie under the name of hypertrophic pulmonary pseudoarthropathy. Not only is this affection, as the name implies, associated with some grave disease of the lungs, such as empyema, but the enlargements met with differ so radically from acromegaly that there is no room for error. It is true that the hands and feet are enlarged, but this enlargement is distinguished by the following peculiarities: in the hands, for instance, the wrists are

very large, the hand itself but slightly enlarged, while the fingers are swollen, especially at their distal phalanges. They are sometimes spoken of as being shaped like drum-sticks. The finger nails, also, are enlarged and frequently curved over the end of the finger like claws or the beaks of birds. The feet present changes similar to those seen in the hands and are not at all like those seen in acromegaly. The face, too, is rounded, while the features are rarely if ever exaggerated. There is no hypertrophy of the lips and tongue.

Osteitis deformans is another affection which is readily differentiated from acromegaly. In contradistinction with the last-mentioned disease, it affects especially the long bones and but rarely the hands and feet, and frequently, because of the involvement of the shafts of the long bones, the latter are much curved and deformed. Secondly, the cranial bones are affected and not especially the features or prominent ridges. Arthritis deformans, again, is an affection which originates in the joints, and the swelling and deformity produced are totally unlike those seen in acromegaly. The metacarpal joints and the phalanges may be enlarged, while the fingers are deflected towards the ulnar side and are often much distorted.

Adiposis dolorosa is an affection in which there is no enlargement of the bony parts whatever, but simply an irregular increase in subcutaneous fatty tissue, the deposit being attended at one time or other by tenderness and neuritic pains.

Treatment.

No satisfactory treatment for the disease has yet been devised. The apparent relation existing between acromegaly and disease of the pituitary gland suggests the relation existing between disease of the thyroid gland and myxœdema and the great efficacy of thyroid preparations in the treatment of the latter disease. Marinesco appears to be the only one who has given the treatment of acromegaly by means of pituitary gland a trial. He has treated three cases of acromegaly by the administration of tablets of desiccated pituitary gland. In two of these cases, a man and woman, there was lessening of the headache and distinct lessening in the size of the extremities. Marinesco thought it probable that the diseased pituitary glands of his patients had undergone diminution in size under the treatment. Certainly treatment by means of desiccated or other preparation of pituitary gland deserves a more extended trial. Unfortunately the remedy is as yet difficult to procure.

The various special symptoms to be combated are headache, somnolence, vertigo, and occasional pains about the trunk and limbs.

The headache can frequently be relieved by the various coal-tar products, phenacetin, antipyrin, or acetanilid. The bromides also appear to be of some service. In cases, however, in which the headache is of great severity recourse must be had to morphine. The somnolence may be combated by coffee, tea, or caffeine, but with indifferent success. General tonics, such as arsenic and iron, may also be employed, but in reality no treatment has any effect upon the disease. Osborne, however, states that tonic treatment, combined with rest, generally causes a cessation in the acute symptoms and a pause in the disease. Mosler suggests the use of ergot. Strychnine, phosphorus, and a milk diet have also been employed at various times. Preparations of the thyroid gland are without effect in acromegaly.

ADIPOSIS DOLOROSA.

Adiposis dolorosa is a disease which is characterized by an irregular, though sometimes symmetrical deposit of fatty masses in various portions of the body, the deposits being preceded or attended by pain. The disease was originally described by the writer in 1892. A case had previously been published by him under the title "A Subcutaneous Connective-Tissue Dystrophy resembling Myxœdema." Subsequently, also, a case was reported by Dr. F. P. Henry under the title of "Myxœdematoid Dystrophy." In addition cases have been studied by Frederick Peterson, B. C. Loveland, Joseph Collins, C. K. Mills, and W. G. Spiller.

Etiology.

Nothing is definitely known of the etiology of this affection save that all of the cases observed, with the exception of one, occurred in women. Secondly, the affection makes its appearance about middle life, or some time subsequently. Perhaps it is significant that some of the cases presented an alcoholic history. In others, again, rheumatism may be a possible factor.

Symptoms.

When seen in the fully developed stage, masses of fatty tissue, variable in size though sometimes exceedingly large, are found distributed over various portions of the body and limbs. Upon examination with the finger it is found that the patient flinches as though the tissue were painful, and pains suggesting a neuritis of terminal nerve filaments are also complained of. At times the tenderness is excessive and the consequent suffering very great. At other times

again the pain is less pronounced, due apparently to the fact that the pathological process, whatever it may be, has passed its most active stage. Occasionally in the same patient a mass of fatty tissue will be found which is no longer painful to pressure, but a history of pain can always be obtained. Very frequently the disease begins by excessive neuritic pains, which make their appearance in limited or diffuse areas of an arm or leg, or possibly in a number of situations at once. Simultaneously with the appearance of the pain, or perhaps subsequently, the part is found to be enlarged, and examination dis-

FIG. 58.—Adiposis Dolorosa (from a photograph).

closes that this enlargement is due to a deposit of fatty tissue. This tissue is boggy, softer than ordinary fat as a rule, and presents at times the pultaceous, worm-like feeling of a variocele. The enormous size attained by some of these swellings is well shown in the accompanying illustrations. Occasionally a nerve trunk is accompanied by a welt-like swelling for some distance of its course. The pains may come on in paroxysms and persist with a high degree of severity for a number of days and then partially subside. The subsidence of the pain may be accompanied by a slight diminution, but never by a disappearance of the swelling. It is a noteworthy fact that the face, hands, and feet have not been involved in any of the cases that have been studied. In addition to the symptoms of fatty deposit and pain which constitute the characteristic features of the disease, we should mention such nervous phenomena as diminished cutaneous sensibility, small patches of anæsthesia, excessive muscular weakness, and at times reaction of degeneration in the muscles. The latter symptom was observed by the writer in one case in the thenar and hypothenar eminences.

Among other symptoms we should also mention hæmatemesis and

epistaxis and excessive menstruation. These symptoms have been repeatedly observed. In keeping with this is perhaps a well-marked purpura, which was observed in one case. Bronchitis and cardiac dyspnœa have been noted in some cases, but the significance of these symptoms is open to question. Headache, herpes zoster, pigmentation of the skin, progressive mental weakness, dementia, should also be mentioned as symptoms that have been observed.

As already stated the pain is variable in degree. It appears also to vary somewhat in character. Thus, as has been stated, it is some-

Fig. 59.—Adiposis Dolorosa (from a photograph).

times paroxysmal, at other times continuous. Sometimes it is described as neuralgic and boring, at other times as burning and scalding. Further, we should bear in mind that in addition to the hands and feet, which are never involved, there may exist various regions upon the trunk and limbs which remain permanently free from the disease. The skin itself shows no change save perhaps occasional pigmentation. It is as a rule soft, white, and flexible, although it is not infrequently very dry. The face is frequently much flushed, especially during the paroxysm of pain. The disease is essentially chronic, extending over many years. It is apt to be progressive. Spontaneous improvement may occur from time to time, though the symptoms never fully subside. The following case will serve as an illustration:

M. G——, aged 51, widow, a native of Ireland and a domestic. Family history negative. Present history as follows:

As a child she had had measles, whooping-cough, and scarlet fever. Menstruation began normally at fifteen. At eighteen she married. Some years later she had an attack of pneumonia but made a good recovery. She had in all seven children and one miscarriage.

Five children died in early childhood, one from cholera infantum, two from measles, one from "congestion of the brain," and the fifth from spasms. The menopause set in abruptly at thirty-five. From this time up to within two or three years her health had continued good. She had undergone some increase in weight, but beyond this nothing worthy of mention could be elicited. Syphilis was denied, as was also alcoholic excess. However, the condition of the patient on several occasions upon her return to the hospital was such as to throw more than doubt upon her denial of alcoholic abuse.

When forty-eight or forty-nine years of age she noticed that her arms were becoming very large. The upper arms and shoulders appeared swollen. The swelling continued steadily to increase and was for about a year unattended by any other symptom.

In November, 1886, she was admitted to the surgical wards of the Philadelphia Hospital for the rupture of a varicose vein of the leg. In the following February she was transferred to the medical wards for a severe attack of bronchitis. Later she had an attack of severe pain and swelling in the right knee, attended by chill and fever. She was treated for rheumatism and promptly relieved. Two weeks after this she complained of a sharp darting pain in the right arm. It began on the outer aspect above the elbow and gradually increased in severity and extent, spreading upwards to the shoulder and neck, and downwards to the forearm and hand. It was shooting and burning in character. She felt at times as though hot water were being poured upon the arm, and again as though the hands and fingers were being torn apart. No rise in temperature was noted. The pain was often paroxysmal, but it was never entirely absent. On June 4th, 1897, she was transferred to the nervous wards of the hospital and came under the care of the writer.

Her appearance at this time was striking. She was a tall, large-framed woman who looked as though she had at one time presented a fine physical development, but she seemed unnaturally broad across the back and shoulders. On removing the clothing an enormous enlargement of these parts was disclosed. The enlargement affected both shoulders, the arms, the back, and the sides of the chest. It was most marked in the upper arms and back, forming here huge and somewhat pendulous masses. It was elastic and yet comparatively firm to the touch, and it was impossible to produce pitting. In some situations it felt as though finely lobulated, and in others, especially on the insides of the arms, as though the flesh were filled with bundles of worms. The skin was not thickened; it did not take part in the swelling, and it was not adherent to the subjacent tissues.

In addition the swelling was very painful to pressure. Pronounced pressure appeared to be absolutely unbearable. The nerve trunks also were exquisitely sensitive, but this painful condition was not by any means limited to them, but permeated the swollen tissue as a whole.

The muscles were not involved in the swelling. The affected parts were, however, quite weak. Examined electrically the muscles of the shoulder and arms yielded a negative result, partly because of the great resistance caused by the intervening tissue. Slight quali-

tative and quantitative changes were noted in the muscles of the forearms, while in the hands distinct reaction of degeneration was noted in the thenar and hypothenar groups, more evident on the right side.

Cutaneous sensibility was much diminished. On the right arm various areas existed in which no response whatever was given to the æsthesiometer. They were large and irregular in shape and very sharply defined, and were present on both the inner and outer aspects. In the finger tips the points could not be at all separated. In the left arm some impairment of sensation was detected on the outer aspect of the forearm, and in the finger tips sensation was decidedly below normal. Sensibility to heat and cold appeared also to have been lessened.

On examining the legs it was found that cutaneous sensibility was distinctly lessened on the right, while showing little or no impairment on the left.

No enlargement was noted at this time in any part of the body save in the arms and shoulders. The face was pale, as were also the mucous membranes. There was, however, a little color in the cheeks, more noticeable at times. Her features were well formed and intelligent. Her hair was dark and fine. Her mind was unimpaired, except that at times she was much abstracted. Sometimes she gave conflicting answers to questions, so that the latter had often to be repeated.

Ten days after her admission to the nervous wards she had a chill, followed by fever and a painful herpetic eruption over the upper portion of the left arm, and anterior portion of the left side of the chest. Some five or six days later another crop of vesicles made its appearance on the back and on the front of the chest.

Nothing further worthy of note occurred until October 13th, when the patient had another severe attack of bronchitis, which was accompanied by much dyspnœa.

In the latter part of the following December it was noted that during one of her paroxysms of pain the swelling of the right arm became more decidedly lobulated. The arm became more sensitive than ever, and on examination hard, cake-like masses were felt, resembling, as the resident physician expressed it, the caking of milk in a breast. This caking or increased lobulated feel was subsequently repeatedly noticed during paroxysms of pain. At this time also she suffered from an attack of pain in the right knee, and in the popliteal space a diffused swelling was felt which exhibited the same nodulated feel as did the swelling elsewhere. It was also very painful.

At various times subsequently paroxysms recurred, during one of which swelling was noticed in the posterior triangles of the neck. The latter seemed later to be permanently fuller than normal. Bronchitis also recurred, accompanied by dyspnœa, and at one time with free expectoration of bloody mucus.

In the following April she experienced an attack of pain of unusual severity. The latter, which involved the right arm and shoulder, right side of trunk and back of neck, now for the first time spread to the face and head. The right side of the face became distinctly swollen, and presented to the touch the same nodulated feel so character-

istic of the swelling in other portions. At the same time the tongue and pharyngeal tissues appeared to become swollen. Her tongue, she said, felt much too large for her mouth. In addition her voice was very hoarse, and she spoke with great difficulty. This condition persisted for upwards of a week, and then slowly subsided. For some time subsequently she spat blood, the source of which was not determined, though it appeared to come from the throat. The reddish color in the cheeks also became more pronounced until it covered the entire forehead like an intense blush. This blush was afterward observed to occur with other paroxysms of pain.

During the summer of 1888 the patient's condition underwent some change. The paroxysms became less frequent and less severe. Hand-in-hand with this improvement sweating became much more marked. However, paroxysms occurred from time to time, and upon one occasion a thick, welt-like swelling, exquisitely painful, was observed extending from the upper and inner angle of the scapula, perpendicularly down the back to very nearly the lumbar region. Upon another occasion, swelling again made its appearance in the right popliteal space, as well as on the inner aspect of the knee. In the latter locality the swelling became permanent, and the tissues presented the same peculiarities as noted elsewhere. Pain also occasionally appeared in the left arm. Prolonged attacks of cardiac dyspnœa occurred every week or two, and apparently independently of bronchitis.

An examination of the eyes by Dr. de Schweinitz revealed contraction of the fields of vision for form and colors, most marked in the left eye. The other special senses, hearing, taste, and smell, appeared to be somewhat obtunded. An analysis of the urine yielded a negative result. A blood count failed to reveal an increase of white blood corpuscles.

Upon a number of occasions the patient vomited blood during paroxysms of pain; upon several occasions this was observed by the writer himself. The quantity could not be accurately estimated, but while it was never large at a single emesis it was constantly brought up in repeated vomiting during an entire night or day.

Measurements were made of this patient at various times, and these showed a steady increase in the bulk of the enlarged parts.

Of late the patient has not suffered as intense pain as formerly. Cardiac dyspnœa, however, is a frequent and distressing symptom. The face is still flushed. Recently shooting pains have appeared in the abdominal region, and examination discloses in this region an extensive deposit of tissue to which the pain is referred. A large longitudinal wheal, especially sensitive, is found in the left lumbar region.

Swelling has also made its appearance over the left hip, and to some extent over the right. The thighs and buttocks do not seem enlarged in proportion, but soft masses are now found on the inner sides of both knees, the right larger than the left, the former more painful to pressure. A small nodule to the right of the scrobiculus is especially painful.

Pathology.

The writer has studied microscopically portions of fatty tissue which he obtained in two of his cases by means of the Duchenne trocar. The fragments revealed connective tissue and fat cells present in varying degree. It was observed that the former was decidedly embryonal in type, the cells being large and fusiform and their nuclei being correspondingly large and prominent. The fat cells for the most part were associated with these connective-tissue cells and occasionally individual fat cells were seen in which the fatty metamorphosis had not been complete. In one of the fragments the writer was fortunate enough to find nerve elements. These had probably been included in the grasp of the trocar by the latter grazing a blood-vessel as the fibres were non-medullated. The latter revealed decided changes. Their connective tissue was denser than normal and they presented an unusual number of nuclei which here and there were aggregated in clusters. In two of the cases studied by the writer autopsies were held, but unfortunately the loss of the specimens prevented the microscopic study of the nervous tissues. In both cases the most striking feature was calcareous infiltration and induration of the thyroid gland.

It is very probable that in this disease we have to do with a connective-tissue dystrophy, a fatty metamorphosis of various stages of completeness occurring in separate regions, or at best unevenly distributed and associated with symptoms suggestive of an irregular and fugitive irritation of the nerve trunks, possibly a neuritis. This view is held by the writer and shared by both Peterson and Collins. That it does not, however, embrace the whole truth is evidenced by such symptoms as the diminished sweating, headache, hæmatemesis, etc., observed in various cases.

Diagnosis.

The affection is readily differentiated from myxœdema by the fact that in the latter disease the face, hands, and feet are involved, and especially by the fact that in myxœdema neuritic pains and pains upon pressure do not occur. For similar reasons it is also readily distinguished from acromegaly. From simple obesity and from lipomatosis perimuscularis circumscripta it is easily differentiated both by the presence of pain and by the irregularity of the distribution of the swelling.

Treatment.

The treatment of adiposis dolorosa resolves itself into the management of special symptoms and the application of general measures. In the writer's cases it has sometimes been necessary to give morphine to control the pain. More frequently, however, the salicylates have proven sufficiently efficacious; for instance, sodium salicylate, oil of gaultheria, and salol. At other times the coal-tar products, antipyrin and phenacetin, have yielded good results. In addition various general measures should be adopted. A diet containing little or no fat-forming food, massage when the pain has sufficiently subsided, sponge-bathing, hydrotherapy in various forms, and galvanism comprise the measures at our disposal. Based upon the observation of the writer with regard to disease of the thyroid gland, Dr. Mills employed in the treatment of one of his cases thyroid extract with, as he reported, very satisfactory results.

In one of the writer's cases dyspnœa was occasionally so great as to necessitate the use of alcohol and digitalis. General principles are, of course, to guide the practitioner in the employment of these remedies.

DISEASES OF THE SPINAL CORD.

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DISEASES OF THE SPINAL CORD.

INTRODUCTION.

The designation "diseases of the spinal cord" is derived from gross anatomy. It is now known to us that the spinal cord consists of four different portions, viz., (1) Processes from cerebral cells, (2) processes from cells which are present in the spinal ganglia and perhaps also in the periphery, (3) cells whose processes extend into the peripheral nerves, and (4) cells which, together with their processes, lie in the spinal cord. It is therefore conceivable that no boundary can be drawn either between the diseases of the spinal cord and those of the brain, or between the latter and those of the peripheral nerves. Besides, there are many affections which involve the brain and the spinal cord at the same time. Thus it happens that various affections are grouped together under the vague designation of "diseases of the spinal cord," while it is to a great extent a matter of choice whether the disease is to be classified or not under this heading. Tabes, for example, is usually regarded as a disease of the spinal cord because the most evident lesions have their seat there. It is, however, really a disease of the entire nervous system, because there are portions of the brain as well as of the peripheral nervous system which are primarily diseased. Multiple sclerosis has been considered in this work among the diseases of the brain, but it might with equal propriety be regarded as a disease of the spinal cord, since both these nervous centres are affected.

Many poisons act injuriously upon the brain and the nerves at the same time, for example alcohol, or it may be only a matter of degree and duration whether changes are found in the nerves alone or in the spinal cord also. To a truly scientific mind, the classification of diseases according to their anatomical seat must appear illogical. Clinical unities correspond with etiological unities, and a strictly etiological classification is perfectly satisfactory. Long usage, however, is hard to overcome, the usual classification is therefore adhered to in this work; but since this is wholly arbitrary, it has not been thought necessary to follow a definite order in the discussion of the several diseases. Following the usual rule in works of this nature, it

would seem proper perhaps to begin with a section on the general etiology and therapy of diseases of the spinal cord, but this is impossible, since the etiology and treatment of these various affections differ widely according to the nature of each. It seems unnecessary also to devote any space to anatomical and physiological details which may be found in any elementary text-book.

INJURIES OF THE SPINAL CORD.

The spinal cord is liable to be injured by sharp or blunt forces. The former variety of injury is principally the result of knife or dagger thrusts. A pointed knife, especially when it is directed from below upwards and somewhat laterally towards the median line, may penetrate between two vertebral arches into the spinal canal from behind without damaging the bone. When the force is very great and the direction of the thrust differs slightly from that above given, more or less serious injury may be inflicted upon the vertebral arches and the blade may penetrate into the canal through this artificial opening. At the same time splinters of bone may be carried along and may in turn produce compression or lesion of the cord.

Perforating or incised wounds of the cord have thus far been observed only in the cervical and upper dorsal regions. This can be due solely to the fact that these parts are more accessible to the hand than the lower dorsal or lumbar region. As regards the latter with reference to an injury to the cord, only the space between the arches of the first and second lumbar vertebræ can come under consideration, for the gap permitting penetration into the cord is particularly large between the arches of these two vertebræ, owing to the horizontal direction of the spinous processes; at this point, should such an accident occur, the cord is liable to be divided in its median portions and perhaps entirely. In cases, however, of a thrust between the vertebral arches in the cervical or dorsal region the division of the cord will almost invariably be merely unilateral, since the vertebral process with its marked downward direction covers the intervertebral fissure in the median line and prevents the penetration of the knife into the opposite side of the cord. Still in some persons the processes of the last two cervical and the first dorsal vertebræ run in a more horizontal direction, and then a total division is possible, as has been observed. In such cases of division of the cord the spinal nerve roots at the level of the thrust are likewise severed; whether it is possible for a knife penetrating into the spinal canal to sever only the roots without producing any lesion of the cord appears to be very doubtful. Aside from the actual knife wound, the effusion

of blood will also damage the cord in these cases. An effusion of blood into the cord in cases of more or less unilateral division will produce at first the symptoms of a complete transverse lesion. In like manner hemorrhages into the meninges may cause at first a more or less complete arrest of the function of the cord at the level of the lesion, while after the lapse of some time only the symptoms dependent on the actually severed portions persist. In the cases which have been described as recoveries from incised wounds of the cord, the injury probably was nothing but such compression by hemorrhages, the knife not having reached the cord itself and perhaps not even the spinal canal.

Injuries of the cord by blunt force are more frequent than those due to cutting instruments. In such cases the force may act directly at the level corresponding to the lesion, the more active part being taken either by the mass causing the injury or by the weight of the human body. The former accident takes place, for instance, in mines or quarries when large masses fall on the back of a laborer, the latter when the subject falls, say from a roof, and strikes with his back the edge of an open door. The writer has observed a case of this kind in a gymnast performing on two horizontal bars, who, intending to strike the second bar from the first with the bend of the knee, struck it with the middle of the back.

Far more frequent than the direct action of the force upon the point of the lesion is an indirect action. Thus spinal injuries are most common in falls upon the head and nucha, especially when the cervical spine is excessively flexed at the same time (as in a fall over the horse's head in clearing a hurdle), or following a heavy blow upon the head. Lesions in the lumbar region, particularly at the sacrum, are associated with a fall on the buttocks.

In most cases of this kind there is probably a lesion of the vertebral column which causes the injury of the cord. In general we may distinguish between luxations and fractures, though both are often combined. Uncomplicated luxations are observed most frequently at the cervical spine, at which point in particular they may at times be diagnosticated, while at the dorsal and lumbar spine (where they also occur) they cannot be distinguished from fractures or fractures combined with dislocation. Of the greatest practical importance are luxations of the lower cervical vertebræ, which are most frequent between the fifth and sixth. Of the vertebræ under consideration it is the upper one that is regarded as dislocated. The luxation occurs at the articulations of the articular processes; usually the lower articular processes of the upper vertebra pass in front of the upper processes of the lower one and become locked (forward luxation). This

luxation may be bilateral (luxation by flexion) or unilateral (luxation by abduction or rotation). In the former case the spinal column above the luxation, and with it of course the head, is inclined forwards; the spinous process of the vertebra below the luxation projects markedly backwards; the vertebral column is fixed not alone by muscular tension but by the patient holding his head firmly so as to avoid all motion. When the luxation is unilateral, the spinous process of the dislocated vertebra is directed towards the luxated side. The head is inclined to the side opposite the luxation and the chin is turned towards the same side. The nuchal muscles are very tense on the luxated side.

Backward luxations of the vertebræ are very rare, and it is even questionable whether they can occur. At the upper cervical vertebræ, however, we meet with forward and backward luxations of the head upon the atlas, and most frequently luxations, likewise forwards and backwards, of the atlas upon the axis. In luxations of the atlas the odontoid process of the axis is probably always broken off; when the luxation is forwards and complete, the arch of the atlas must also be broken off. Luxations by rotation of the atlas may likewise occur.

Fractures of the vertebral column may affect the bodies of the vertebræ, the arches, the spinous processes, or the articular and transverse processes. Fractures of the bodies are most frequently the result of indirect force, those of the arches of direct force; the articular processes break mainly with luxations. The vertebral bodies may be completely comminuted; sometimes, though rarely, splinters may be displaced into the vertebral canal where they of course injure the cord. Vertebral fractures, too, are most common in the cervical and upper dorsal portions; next in order are the lower dorsal and the first lumbar vertebræ. They may be confined to a single vertebra or involve several; the former occurrence is most frequent in the lumbar portion, the latter in the upper dorsal region.

A differentiation between fractures and luxations is but rarely possible, especially as both conditions are often combined; it is most likely to be made in the cervical portion and in that case crepitation would indicate fracture, though we should take care not to produce it. Fractures of arches and spinous processes are more readily recognized. Marked projection or depression of a dorsal spinous process indicates a luxation.

Sometimes there is neither a luxation nor a fracture of the vertebræ but merely a diastasis by rupture of the ligamentous apparatus or of the intervertebral discs, though as a rule all these conditions are associated with each other. Hemorrhages in considerable amount into the spinal canal are nearly always present. In very severe cases

the external integument is also injured so that there is an open wound of the spinal canal or cord.

Midway between sharp and blunt forces acting upon the spinal cord are lesions caused by small projectiles, *e.g.*, revolver bullets, which may produce only slight injuries of the vertebral column; larger projectiles of course must seriously damage the latter before they strike the cord. The lesions produced in the cord by the above-mentioned injuries of the vertebral column may vary greatly. In some cases it is not wounded at all, particularly in unilateral luxations and in fractures of the vertebral arches or of the spinous processes. The lesions of the cord are also slight when they consist essentially in a compression by an intervertebral hemorrhage. In the most serious cases there are true breaches of continuity of the cord which may, for instance, be torn in excessive flexion of the vertebral column or severed by a pointed fragment of bone. Between these are all possible transitions—complete contusion of the cord at the level of the lesion so that the cord is represented merely by a sac containing detritus, partial contusions, and more or less total suffusion of the cord with blood. When infection is made possible by an external injury, *e.g.*, in gunshot wounds, such lesions are ultimately complicated with purulent meningitis or the formation of abscesses in the cord.

In quite a number of lesions caused by the direct or indirect action of force upon the vertebral column, serious injuries may be present in the cord without any material alterations being discoverable in the bones at the autopsy. In a few of these cases there may have been at the time of the accident some slight displacement of the vertebræ upon each other or a luxation with contusion of the cord; but spontaneous reduction must have followed at once so that no evident structural disturbance would be found in the vertebral column.

Unquestionably, however, there are cases—and they are not so rare as was formerly believed—in which even at the time of the accident the vertebral column remained intact, and a disintegration and hemorrhage in the cord resulted either from strong traction or violent contusion. These cases belong properly in the class of traumatic hæmatomyelia. In the milder cases the hemorrhage is confined to the whole of the gray substance or a portion of it for a long distance (tubular hemorrhage); in the severe cases the entire cross section may be permanently injured. In such cases it is hardly ever possible to exclude a lesion of the vertebral column during life.

Pathological Anatomy.

The alterations of the cord in cases of injury are at least qualitatively always the same and relatively simple. There is invariably more or less pronounced destruction of medullary substance with all its consequences, and finally, when the duration is sufficiently prolonged, cicatrization. In simple knife thrusts the destruction, especially in the longitudinal axis of the cord, may not be extensive. In recent cases we shall find, usually confined to one side of the cord, a short focus of so-called red softening, in which the point of the knife is sometimes still embedded, the histological elements of which consist of red and scanty white blood corpuscles, broken-down myelin, disintegrating ganglion cells, and blood-vessels gorged with blood or ruptured. The lesion very gradually merges into the surrounding healthy tissue. In the transitional zone signs of reaction appear at first in the shape of profuse proliferation of in part newly formed blood-vessels, and swelling and proliferation of the neuroglia, with the presence of granular cells. The latter penetrate into the disintegrated tissue. At this point there is a gradual resorption of the destroyed nerve tissue and also a proliferation of the neuroglia, which is usually in a state of better preservation, perhaps of some connective tissue and of the blood-vessels, so that at last the nerve tissue is replaced by pure cicatricial tissue whose origin is betrayed merely by scanty remnants of ganglion cells, some disintegrated myelin, and residues of hemorrhage. This cicatrix remains permanently; a reunion of the divided nerve fibres does not occur in the spinal cord even when the cicatrices are very short, and while the posterior nerve roots, which have been severed and detached from the cord, may grow again for some distance into the cord, they do not unite with their central cut ends. The hemorrhages into the meninges may disappear or they may become organized and remain as a kind of induration between the meninges. As to possible ascending and descending degenerations, the reader is referred to a later paragraph. In extensive destructions, such as occur with luxations and fractures, the process is essentially the same as stated above. When the contusion has been extreme, the pia, whose continuity is nearly always preserved, contains at the point of the lesion only a mass of detritus which at first is usually stained red by a considerable admixture of blood. When the dislocation of the vertebræ with the resulting narrowing of the spinal canal persists, the cord will of course be thinned and compressed even in the first stage of the injury; after the dislocation has been reduced, the cord may be swollen. Subse-

quently the course of the alterations at the point of the lesion and its vicinity, in these cases of extensive injury, is exactly the same as with the smaller lesions, *i.e.*, resorption of the disintegrated and hemorrhagic medullary substance; proliferation of glia, connective tissue, and blood-vessels within and around the focus; ultimately contraction and sclerosis of the entire region into a connective-tissue cicatrix. Of course this cicatrix after severe contusions will occupy a much larger surface, and the cord may then be thinned, ribbon-like, or be changed to a loose sac, consisting chiefly of pia and a cicatricial tissue containing usually numerous blood-vessels, some ganglion cells, blood pigment, and remnants of myelin.

Hemorrhages into the meninges associated with these extensive lesions undergo precisely the same changes as those occurring in mild cases.

Above and below the cicatrix we find ascending and descending degeneration. When the entire cross section is destroyed we observe in an ascending direction a degeneration of the posterior columns, and especially of the columns of Goll and Gowers and the direct cerebellar tracts, of the margins of the anterior columns, and for some distance also of the pyramidal tracts; in a descending direction a degeneration of the pyramidal tracts, of the margin of the entire antero-lateral tract, of the comma-shaped tracts in the posterior columns, and a tract in the latter which passes first at the posterior periphery of the cord, then as an oval field beside the posterior septum, and reaches as far as the filum terminale (Hoche). When the transverse section of the cord is only partially destroyed, these secondary degenerations of course are present to a corresponding degree.

Along with these system-degenerations we very often find, particularly with traumatisms, though not exclusively with them, chiefly in the centre of the posterior columns and in the posterior horns above and below the total lesion, irregularly scattered, degenerated, usually round spots, which appear light after hardening in chromic acid—the so-called disseminated patches. Examining these in serial sections we find that they are cylindrical foci of degeneration extending often over quite a number of segments. The degenerated cylinder is frequently completely detached from the surroundings, lying in the cord like a necrotic piece of bone does in its bed. Finally it may disappear altogether so that there is left an irregular cavity extending over several segments. These disseminated patches probably represent a direct necrosis of the implicated regions; this is shown by the fact that they stain little or not at all; they have a delicate reticulated structure (the network is formed of glia fibres and the finest blood-

vessels) and usually contain within the meshes remnants of nerve fibres and blood pigment. In the adjoining non-necrotic cord, eventually also at the margin of the cavities, we find numerous granular cells.

Of course the lesion need not necessarily involve the whole cross section of the cord. As a rule the central portions, the gray substance, suffer most, and even in very severe lesions some stretches of moderately preserved nerve substance frequently remain immediately under the pia. Quite interesting and of practical importance is also the condition of the spinal nerve roots. Those issuing from the destroyed segments are of course degenerated and atrophy occurs also in the muscles supplied by the anterior roots. On the other hand it is a matter of common observation that in otherwise very serious lesions those nerve roots which merely pass by the injured cord to reach their point of emergence from the spinal canal, and which, for instance, in the dorsal portion of the vertebral canal are derived from segments considerably above the seat of the injury, are well preserved; while *a priori* it would be assumed that these roots, lying as they do particularly close to the injured cord, would be more liable to be affected than the cord. In general, however, the roots are more resistant than the cord, and this not only with reference to traumatisms; to be sure their power of resistance does not go beyond a certain limit; when the trauma is very forcible they likewise are torn.

In extremely severe cases, in excessive flexion or marked diastasis of the vertebral column, the cord may be torn, leaving between the two ends a hiatus several centimetres in length, which is filled with blood. Under the microscope the two ends of the cord present the picture of the most severe contusion.

When the nature of the injury furnishes an opportunity for infection, such cases may be attacked by purulent meningitis or more rarely by abscess of the cord. Both of these conditions, however, occur also secondarily from a severe bedsore or possibly from cystitis. In cases in which the cord is injured while the bones are intact, the clinical and anatomical condition is that of hæmatomyelia. The hemorrhage in these cases is generally located in the gray substance but extends as a tubular hemorrhage often over long distances; in some instances it affects only a portion of the gray columns, a posterior or an anterior horn. For further details the reader may consult the section on hæmatomyelia.

Symptoms.

For the sake of simplicity we shall discuss here first the symptoms of severe contusion of the cord, caused by blunt force and involving marked injury of the vertebral column. Let us assume that we have to deal with the most frequent accident, with a considerable though not quite complete interruption of the transverse section involving one segment of the cord or a number of them. The symptoms manifested may be divided into those produced by the segments directly injured, and those due to the interrupted conduction below the lesion. With reference to both, the following may be stated as of general application: As regards segmental symptoms, a differentiation between root and cord symptoms, such as we are familiar with in slow compression of the cord, is usually impossible in traumatism. In slow compression the lesion of the roots may in typical cases long precede those of the cord, and both phases may manifest themselves by special symptoms; in acute traumatism both the cord lesion and any possible root lesions occur at the same time. Since the symptoms in injury of a spinal segment or of the roots springing from it are exactly the same, and in most cases of spinal lesion only those roots become diseased which spring from the injured segment, but not the roots of higher segments passing beside the injured segment to their points of emergence from the spinal canal, it is easily understood that in serious contusions root and cord symptoms coincide. Only in isolated cases of extremely grave vertebral injury a portion of the symptoms—those whose location is highest—may be referred to a root lesion *per se;* we shall see hereafter when this is so and that this condition is easily recognized.

In the region supplied by the injured segment, in supposedly more or less total transverse lesions, a complete cessation of the cord functions must result. The muscles dependent upon the segment will be affected with a flaccid paralysis, they will undergo atrophy, and will exhibit the reaction of degeneration. Sensibility must be completely abolished in the same regions. The tendon and cutaneous reflexes whose paths run through the injured segments must vanish. For reasons to be discussed below, all these symptoms, especially the atrophy and the electrical disturbances of the muscles, will be more distinct when several segments are affected.

The symptoms of interrupted conductivity below the lesion find expression in disturbances of motility, of sensibility, of the reflexes, and of the sphincters, and in trophic derangements. All muscles whose nuclear fields lie below the transverse lesion are paralyzed, and this

paralysis is always spastic and after some time leads to a contracture in extension or flexion of the paralyzed limb. The sensibility is lost up to the level of the cutaneous region supplied by the injured segment; a zone of hyperæsthesia sometimes presents itself above the anæsthesia as a segmental symptom. The tendon reflexes, when the transverse section is not completely interrupted, are increased so far as their reflex arc lies below the lesion, and a clonus of the patellar and Achilles tendons forms rapidly. These spastic manifestations are frequently so strong that even very slight movements of the legs induce shaking motions of all the muscles (the spinal epilepsy of Brown-Séquard). The cutaneous reflexes may also be increased below the point of the lesion, at all events they are present in most cases. As the disturbances of the sphincters may vary greatly, it will be better to discuss them in connection with the symptom complexes occurring in injury of the several segments of the cord. Often there is first retention of urine, later incontinence. Bedsores occur particularly when the disturbance of sensibility is total; cystitis develops after marked paralysis of the bladder. When the transverse lesion is almost total, the secretion of sweat in the entire region below it is arrested; in the paralyzed legs we often find œdema and increased temperature of the skin. This much can be said in general about the symptoms in severe transverse lesions of the cord. Otherwise the symptoms of course will vary much, particularly in their extent and grouping, according to the site of the lesion with reference to the longitudinal axis of the cord. The higher the situation of the lesion the larger are the regions in which nerve conduction is interrupted, and the more extensive therefore will be the disturbances which have been briefly enumerated above. The lesions may be roughly divided into those of the upper cervical cord, those of the cervical enlargement (subdivided into those of the upper and lower half), those of the dorsal cord, and those of the lumbar and sacral portions. Besides there are the lesions of the uppermost cervical cord and those of the cauda equina.

In lesions of the dorsal cord (in this connection the symptoms of the injured segment and those of the conducting paths are enumerated together; when feasible first the former, then the latter) the following symptoms are found: Girdle pain; a hyperæsthetic zone corresponding to the level of the lesion; spastic paralysis of the legs, usually in extension, more rarely in flexion; patellar and ankle clonus; often marked so-called spinal epilepsy; anæsthesia up to and including the diseased segment; always preserved and often increased cutaneous reflexes; variable disturbances of the vesical functions, difficult micturition with violent tenesmus, sometimes involuntary evacuation,

frequently retention of urine or paradoxical ischuria; constipation, subsequently bedsores, cystitis, etc.

When the lesion is located in the lower portion of the cervical enlargement, possibly pains and zones of hyperæsthesia are found only in the ulnar part of the arm. We find also atrophic paralysis of the small muscles of the hand, of the flexors and extensors of the fingers and eventually of the hand, later with reaction of degeneration. Anæsthesia of the trunk extends to the level of the second rib in front and of the spine of the scapula behind; in the arm, it implicates only the ulnar half. Contraction of the pupils and of the palpebral fissures exists, and there is paralysis of the muscles of the trunk and legs. The condition of the reflexes and sphincters is the same as in injury to the dorsal cord.

In lesions at this point we often observe, as pointed out by Thorburn, a peculiar position of the arms; at the shoulder they are abducted, at the elbow flexed and pronated. This is due to the fact that the muscles required for this position, which are mostly derived from the fifth cervical root, are not involved in the paralysis in these cases and are sometimes perhaps even in a state of slight contracture.

When the lesion affects the upper part of the cervical enlargement, the forearm and hand present a spastic paralysis, the upper arm and shoulder an atrophic paralysis. Pains, if present, are located chiefly in the shoulder. The anæsthesia affects the arms over their entire extent. Otherwise all the conditions are the same as in lesions of the lower half of the cervical enlargement. The state of the pupils and palpebral fissures is still undetermined.

In injury to the upper half of the cervical cord a spastic paralysis of all four extremities would occur and the muscles concerned in the movements of the head would also be implicated. The anæsthesia will involve also portions of the nuchal and cervical regions. The diaphragm must likewise be paralyzed. In luxations of the uppermost cervical vertebræ, besides the above-described abnormal position of the head and neck, pain in the occiput and stiffness of the nucha are also present. If the cord is involved in any degree, all the spinal muscles and eventually also the hypoglossi are paralyzed. Unless aid is immediately rendered, death is bound to occur soon. In lesions of any part of the cervical enlargement, erection of the penis is very frequent or there is at least some turgidity of the member which increases with any manipulation, for instance, during catheterization. Not rarely pyrexia has been observed in addition, for which there has been no other explanation.

When the upper portion of the lumbar enlargement of the cord is injured, there may be occasionally flaccid atrophic paralysis in the

paralysis is always spastic and after some time leads to a contracture in extension or flexion of the paralyzed limb. The sensibility is lost up to the level of the cutaneous region supplied by the injured segment; a zone of hyperæsthesia sometimes presents itself above the anæsthesia as a segmental symptom. The tendon reflexes, when the transverse section is not completely interrupted, are increased so far as their reflex arc lies below the lesion, and a clonus of the patellar and Achilles tendons forms rapidly. These spastic manifestations are frequently so strong that even very slight movements of the legs induce shaking motions of all the muscles (the spinal epilepsy of Brown-Séquard). The cutaneous reflexes may also be increased below the point of the lesion, at all events they are present in most cases. As the disturbances of the sphincters may vary greatly, it will be better to discuss them in connection with the symptom complexes occurring in injury of the several segments of the cord. Often there is first retention of urine, later incontinence. Bedsores occur particularly when the disturbance of sensibility is total; cystitis develops after marked paralysis of the bladder. When the transverse lesion is almost total, the secretion of sweat in the entire region below it is arrested; in the paralyzed legs we often find œdema and increased temperature of the skin. This much can be said in general about the symptoms in severe transverse lesions of the cord. Otherwise the symptoms of course will vary much, particularly in their extent and grouping, according to the site of the lesion with reference to the longitudinal axis of the cord. The higher the situation of the lesion the larger are the regions in which nerve conduction is interrupted, and the more extensive therefore will be the disturbances which have been briefly enumerated above. The lesions may be roughly divided into those of the upper cervical cord, those of the cervical enlargement (subdivided into those of the upper and lower half), those of the dorsal cord, and those of the lumbar and sacral portions. Besides there are the lesions of the uppermost cervical cord and those of the cauda equina.

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frequently retention of urine or paradoxical ischuria; constipation, subsequently bedsores, cystitis, etc.

When the lesion is located in the lower portion of the cervical enlargement, possibly pains and zones of hyperæsthesia are found only in the ulnar part of the arm. We find also atrophic paralysis of the small muscles of the hand, of the flexors and extensors of the fingers and eventually of the hand, later with reaction of degeneration. Anæsthesia of the trunk extends to the level of the second rib in front and of the spine of the scapula behind; in the arm, it implicates only the ulnar half. Contraction of the pupils and of the palpebral fissures exists, and there is paralysis of the muscles of the trunk and legs. The condition of the reflexes and sphincters is the same as in injury to the dorsal cord.

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When the upper portion of the lumbar enlargement of the cord is injured, there may be occasionally flaccid atrophic paralysis in the

distribution of the crural and obturator nerves, with absence of the patellar reflex. The anæsthesia occupies the whole of both legs. Pains, if present, are found in the distribution of the crural nerve. The disturbances of the bladder and rectum may be the same as in lesions of the dorsal cord. In contusion of the sacral cord we find atrophic degenerative paralysis in the muscles of the leg and foot, possibly also in the flexors of the thigh. The remaining muscles of the thigh are normal; the patellar reflex may be preserved, the Achilles tendon reflex is absent. The anæsthesia involves the foot and posterior surface of the thighs and legs. The bladder is completely paralyzed and contains only so much urine as the sphincter vesicæ can retain by its elasticity; when this amount is exceeded, the urine dribbles away. The bladder can be emptied by expression. Restriction of the injury to the sacral plexus is to be expected only when there is an isolated lesion of the cord, the lumbar roots passing by the sacral cord remaining intact. When all of the lumbar enlargement is destroyed—a condition usually observed in injury of the lower dorsal and upper lumbar vertebræ—we find atrophic paralysis of the whole of the legs, absence of the tendon reflexes, anæsthesia extending upwards slightly above the flexure of the thigh, and paralysis of the bladder as in contusions of the sacral cord. In these cases bedsores generally develop with special facility and spread rapidly. When the conus medullaris is alone injured, there is anæsthesia of the perineum, of the genitals, and eventually also over a median strip at the posterior surface of the thigh, together with paralysis of the bladder and impotence.

When the lesion involves the spine below the second lumbar vertebra, only the cauda equina can be injured; the symptoms in this case are usually the same as in lesions of the sacral cord, rarely as in those of the entire lumbar enlargement. As a rule the pains are more violent than in injuries of the corresponding segments of the cord, and the paralysis is not so symmetrical on the two sides as it is in cord lesions.

This completes the rough description of the symptoms caused by lesions at the various levels of the cord, particularly as regards their extent and grouping. But it is especially in injuries of the cord that such a description in bold outlines proves insufficient, above all with our present demands upon diagnosis with reference to a possible causal treatment. In every single case we must exactly determine the extent of the paralyses and anæsthesias, and this applies above all to the upper limits of these two symptom groups, as we demonstrate at the same time the upper limit of the lesion in the cord. This will convince us that chance does not govern here, that

every definite level of the lesion determines a positive limit for the disturbances of sensibility, and that a certain number of muscles are paralyzed, while other groups are preserved, the latter becoming less in proportion as the level of the lesion rises. This grouping corresponds in detail to what we know thus far regarding the localization of the functions of the several segments of the cord. Since a lesion causes no clinical symptoms above its upper level, although it produces ascending degeneration, the highest paralyzed muscles must invariably correspond to the upper limit of this lesion and the muscle groups supplied from above this point will not be paralyzed. Thus, for instance, with a lesion situated in the sixth cervical segment, the paralysis will affect all the arm muscles except those depending upon the fifth cervical segment, namely, the deltoid, biceps, brachialis internus, and supinator longus. The same remark applies to sensibility; when the upper end of the lesion reaches, say, the seventh cervical segment, the entire ulnar half of the arms will be anæsthetic, while the radial half will have normal sensibility.

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD.

Segment.	Muscles.	Reflexes.	Cutaneous Sensation.
First cervical	Rectus lateralis. Rectus capitis. Anticus and posticus. Sternohyoid. Sternothyroid.		
Second and third cervical	Sternomastoid. Trapezius. Scaleni and neck. Diaphragm.	Inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex and neck.
Fourth cervical	Diaphragm. Deltoid. Biceps. Coracobrachialis. Supinator longus. Rhomboid. Supra- and infraspinatus.	Dilatation of the pupil produced by irritation of neck (fourth to seventh cervical).	Back of neck. Shoulder, upper surface. Outer surface of arm. Anterior portion thorax as far as the second rib.
Fifth cervical	Deltoid. Biceps. Coracobrachialis. Brachialis anticus, Supinator longus. Supinator brevis. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	Scapular (fifth cervical to first dorsal). Tendon reflexes of the corresponding muscles.	Back of shoulder and arm. Outer side of arm and forearm.
Sixth cervical	Biceps. Brachialis anticus. Extensors of the hand and fingers. Pectoralis (clavicular part). Serratus magnus. Triceps. Pronators.	Tendon reflexes of the extensors of the arm and forearm. Posterior wrist (sixth to eighth cervical). Tapping tendon causes extension of hand.	Outer side of forearm. Back of hand, radial distribution.

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Fourth cervical	Diaphragm. Deltoid. Biceps. Coracobrachialis. Supinator longus. Rhomboid. Supra- and infraspinatus.	Dilatation of the pupil produced by irritation of neck (fourth to seventh cervical).	Back of neck. Shoulder, upper surface. Outer surface of arm. Anterior portion thorax as far as the second rib.
Fifth cervical	Deltoid. Biceps. Coracobrachialis. Brachialis anticus. Supinator longus. Supinator brevis. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	Scapular (fifth cervical to first dorsal). Tendon reflexes of the corresponding muscles.	Back of shoulder and arm. Outer side of arm and forearm.
Sixth cervical	Biceps. Brachialis anticus. Extensors of the hand and fingers. Pectoralis (clavicular part). Serratus magnus. Triceps. Pronators.	Tendon reflexes of the extensors of the arm and forearm. Posterior wrist (sixth to eighth cervical). Tapping tendon causes extension of hand.	Outer side of forearm. Back of hand, radial distribution.

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD.—*Continued.*

Segment.	Muscles.	Reflexes.	Cutaneous Sensation.
Seventh cervical.	Triceps (long head). Extensors of hand and fingers. Pronators of hand. Flexors of hand. Subscapular. Pectoralis (costal part). Latissimus dorsi. Teres major.	Palmar (seventh cervical to first dorsal). Stroking palm causes closure of fingers.	Radial distribution in the hand. Median distribution in the hand.
Eighth cervical.	Flexors of hand and fingers. Intrinsic hand muscles	....	Ulnar area of hand, back and palm; inner border of forearm. (Internal cutaneous, ulnar.)
First dorsal..	Extensors of thumb. Intrinsic hand muscles Thenar and hypothenar muscles.	....	Ulnar area.
Second to twelfth dorsal.	Muscles of back and abdomen. Erectores spinæ.	Epigastric (fourth to seventh dorsal). Abdominal (seventh to eleventh dorsal). Vasomotor centres. Second dorsal to second lumbar.	Skin of chest, back, and abdomen. Upper gluteal region.
First lumbar.	Ileopsoas. Sartorius. Abdominal muscles.	Cremasteric (first to third lumbar).	Skin over groin and front of scrotum.
Second lumbar.	Ileopsoas. Sartorius. Flexors of the leg (? Remak). Quadriceps femoris.	Patellar tendon (second to fourth lumbar).	Outer side of thigh.
Third lumbar.	Adductors and inward rotators of thigh. Flexors of thigh.	....	Front and inner side of thigh.
Fourth lumbar.	Adductors and abductors of thigh. Tibialis anticus. Flexors of leg (? Ferrier).	Gluteal (fourth to fifth lumbar).	Inner side of thigh, leg, and foot.
Fifth lumbar.	Outward rotators. Flexors of leg (? Ferrier). Flexors of foot. Peronei. Extensors of toes.	....	Back of thigh and outer side of foot.
First and second sacral.	Flexors of foot and toes. Peronei. Small muscles of foot.	Plantar (fifth lumbar to second sacral).	Back of thigh, outer side of foot.
Third to fifth sacral.	Muscles of the perineum.	Achilles tendon. Vesical centre. Anal centre.	Sacral region, anus, perineum, genitals.

In the appended table (after Allen Starr and Edinger, with a few slight modifications), the muscles, reflexes, and cutaneous regions dependent upon the several spinal segments are enumerated in order. This table is in general quite reliable. In a concrete case, therefore,

the limit of the anæsthesia and the extent of the muscular paralysis having been accurately determined, we can ascertain from the table which are the highest segments injured. Below this level everything is paralyzed and anæsthetic. In this connection it should be noted that the data for this table have been gathered in the main from traumatic cases and that we are indebted to Thorburn for the most accurate observations; of special importance were the cases in which, probably by reason of hemorrhage or an advancing œdema, a gradual rise of the lesion could be observed for a few days following the traumatism. The table shows that the conditions are quite simple at the dorsal cord; they are considerably more complicated at the cervical and lumbar enlargements, and particularly at the latter point many facts are still obscure and in need of further elucidation.

In making use of the table and diagram it is necessary to observe some precautions, which, however, have been at least in part noted therein. We are indebted to Sherrington for the positive proof that in no instance does a single segment or a single root alone supply a definite muscle or a definite region of the skin, but that a number, at least three segments and roots, participate. A complete paralysis of the respective muscle and an absolute anæsthesia of the respective cutaneous region occur only when all the corresponding segments are destroyed. This rule holds good especially in the development of marked atrophy and of the reaction of degeneration in the paralyzed muscles. Therefore, according to the appended table, in complete paralysis of the deltoid we must assume an implication of the fourth cervical segment, and in paralysis of the biceps an implication of the fifth

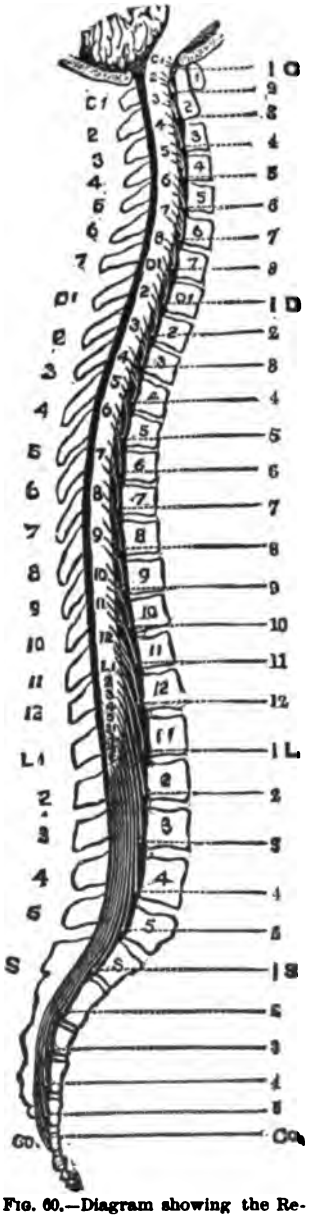

Fig. 60.—Diagram showing the Relations of the Spinous Processes to the Bodies of the Vertebræ and of these to the Points of Origin of the Spinal Nerves (Gowers).

inflammation will manifest itself by corresponding pain, rigidity of the back and nucha, and eventually by cerebral symptoms. An abscess in the region destroyed by traumatism can produce no new symptoms. In these cases fever will set in, which otherwise is very rare in cord lesions, aside from those located high in the cervical cord, or which is due to complications such as cystitis, pyelitis, and hypostatic pneumonia.

Course, Duration, and Termination.

The process in the cord is not always, even aside from true infections, complete with the cessation of the traumatism. On the contrary, in most cases probably a capillary hemorrhage or an œdema spreads upwards and downwards for some time after the injury. Since in approximately total transverse lesions such an eccentrically extending hemorrhage or œdema cannot produce new clinical symptoms below the injured cross section, the impression is gained in these cases that the morbid process advances only upwards. In such cases as have been observed particularly by Thorburn, one segment after another above the lesion is gradually attacked; at first perhaps only the hand muscles are paralyzed, then those of the forearm, finally those of the upper arm and shoulder. The limit of the anæsthesia extends in like manner by degrees higher and higher. Formerly these processes were designated as progressive so-called traumatic myelitis; but in uncomplicated cases there is no ground for assuming the presence of an inflammation, the after-hemorrhage and the œdema explain everything. If the patient continues to live, these symptoms which are not directly dependent upon the contusion of the cord again recede.

In the beginning the morbid picture of the cord lesion, at least in grave contusions, is complicated by the general shock, which in itself sometimes causes the death of the patient. To the same condition was formerly also ascribed the absence of the tendon reflexes soon after the injury, even when this was located in the dorsal or cervical cord, especially in cases in which the reflexes reappeared in a few days and then rapidly increased. It is just as likely, however, that in these cases there was at first a complete interruption of conduction in which naturally the tendon reflexes were absent, and that this retrogressed so as to be only partial by the end of a few days.

Death is the most frequent termination of such serious injuries of the cord. When a lesion involves the cervical cord close to the base of the skull—for instance, in luxation of the atlas—and reduction cannot be at once effected, or if the injury to the cord is very serious

death will rapidly and sometimes immediately follow the accident. This is the case also when the region of the phrenic nuclei is directly involved.

Death by asphyxia occurs early, usually a few days after the injury, when the seat of the lesion is high—for instance, in the upper dorsal cord—and the secondary œdema or the after-hemorrhage in its upward spread involves more and more of the centres for the auxiliary muscles of respiration and finally also the phrenic nerve. Relatively as rapid is the effect of an infection with meningitis and abscess formation or of some other intercurrent disease, especially pneumonia. In the great majority of cases, however, the patients survive for some time after the injury and do not succumb until weeks or months later to various complications, most frequently bedsores, then cystitis, pyelitis and nephritis, or pneumonia, finally also general marasmus. These secondary morbid phenomena of course predominate in the later periods of the disease, after the recovery from shock. Along with them all the indirect sequelæ of the traumatism—muscular atrophies, contractures, etc.—may have sufficient time to develop.

A fatal issue therefore is nearly inevitable in all cases of grave injury of the cord, and the only question is as to the time which intervenes between the lesion and the lethal termination. In general it may be said that life is jeopardized the more seriously and rapidly the higher is the location of the lesion and hence the more extensive are the functional disturbances caused by it. A lesion of the lumbar cord therefore is at first least dangerous to life, but subsequently it becomes more so than one involving the dorsal cord, owing to the disturbances of the bladder which are usually serious. Lesions of the cervical cord are nearly always rapidly fatal. Injuries of the cauda equina *per se* do not endanger life.

The prognosis depends also upon the extent of the lesion in the cross section. Total transverse lesions in which every function below the injury has ceased are more rapidly and more surely fatal than partial lesions, be the intact portion of the cross section ever so small. Still, however common a fatal termination may be in grave traumatisms of the spinal cord, it is not absolutely inevitable even in the most serious cases. Thus, for instance, Egger has described a case of total transverse lesion in the upper dorsal cord in which the patient lived for eleven years. Improvement of course did not occur in this case. When, however, the actual lesion of the cross section is partial from the first, the prospects as to life are of course much better, and a number of patients have been saved, for instance, after unilateral lesions caused by knife thrusts. Even marked improvement may occur in such cases, and all the symptoms due to slight

sanguineous or œdematous infiltration of the cord may retrogress. As a rule in such cases of lesion of the dorsal cord the sensory disturbances and those of the sphincters may retrogress and a spastic paralysis persist; in lesions of the lumbar cord a permanent flaccid paralysis in more or less extensive muscular regions would remain. A termination in complete recovery has also been reported in isolated cases of cord lesions. Nowadays, however, we do not believe in recovery after true lesions of the cord, and in such cases there was probably only a hemorrhage into the spinal meninges with compression.

Diagnosis.

The diagnosis of a lesion of the spinal cord *per se* is in most cases easy. Given the above-cited causes—knife thrust, gunshot wound, direct or indirect traumatism by blunt force—followed by more or less extensive paraplegia with perfect exemption of the brain, the diagnosis of a cord lesion is probably always imperative. In these cases we do not even require the demonstration of the vertebral injury, though the latter will materially strengthen the diagnosis.

It will often be difficult if not impossible to differentiate a hæmatorrhachis with compression of the cord from a true traumatism. With hemorrhage into the spinal meninges the pains and the rigidity of the back are said to be more marked than with actual contusion of the cord; but these differences will be of little avail, since a lesion of the cord will rarely occur without hæmatorrhachis. The final termination in complete or almost perfect recovery will always point to a hemorrhage into the meninges. Moreover, hysterical and especially traumatic-hysterical paralysis may occasionally cause confusion for a short time. In the first place, however, traumatic hysteria has usually a period of incubation; it does not occur until days after the injury. Secondly, monoplegias are more frequent in it than paraplegias. Thirdly, the sensory disturbances do not correspond to those observed in cord lesions, but are distributed to other points determined by psychical conditions. Thus, in the hysterical, a segment of a member is an entity; if the whole arm is paralyzed it is also quite anæsthetic; if it is paralyzed only to the elbow the anæsthesia likewise reaches only to the same point, etc. Finally, in hysteria there is an absence of all serious complications, such as bedsores, cystitis, and marasmus.

With reference to certain therapeutical questions, it would be of special importance if we could say in a concrete case whether the paralyses present depend upon a persisting compression of the cord by the displaced spinal column or by isolated splinters of bone, or

whether they are due solely to the destruction of the cord caused by the traumatism. It should be stated in the first place that even the gravest contusions with complete and permanent interruption of the cross section may occur without lesion of the vertebral column, and furthermore that while at the moment of the accident the cord may have been bruised by a considerable displacement of the body of the vertebra, this displacement may have been at once spontaneously reduced. A continuous contusion is most probable of course with the persistence of a marked deformity of the spinal column, but even then it is not necessarily present, since the vertebral canal leaves comparatively much room to the cord and the latter may adapt itself to pronounced deformities of the spinal column. Obviously in these cases likewise, when the displacement occurs rapidly, the cord at the point of the dislocation may be not simply compressed but may be disintegrated. On the other hand, lesions of the spinal column, particularly of the bodies of the vertebræ, are often not at all demonstrable during life, and, therefore, in the absence of a perceptible deformity we can never assert that none of the vertebral bodies or a splinter from them is pressing on the cord. In short, in not a single instance can we really assert a continuous compression of the cord by bone nor can we quite exclude it.

A diagnosis of injury of the cord alone is not sufficient, for it is essential to know at what level the cord is injured, in other words to make the segmental diagnosis. Of course this is easy when a distinct deformity of the spinal column is present. In that case we need but ascertain opposite which spinal segment the injury in question is situated, and on careful determination of all the morbid symptoms of the patient we shall always find that the symptoms discovered correspond to the injured point. We have seen above that in such cases of injury of the dorsal cord the limit of the upper level of the anæsthesia, when the transverse lesion is total, usually lies considerably below the point of spinal deformity—the difference is about three vertebral spines—and we have also found the explanation of this variation which at first sight appears striking. As this is a matter of importance, we may repeat here that it is due in the first place to the fact that in traumatism, as a rule, only the single spinal segments and the roots emerging from them are injured, but not the roots derived from higher segments and merely situated alongside the damaged cord; secondly, to the fact that, in the dorsal region in particular, the roots from their origin to their emergence from the spinal column have to pass a long way, corresponding to the height of two vertebral bodies; and thirdly, to the fact that while every cutaneous region is chiefly supplied by a special

root, the immediately adjoining upper and lower roots participate in the innervation (the upper alone here enters into the question), and not until all these roots are destroyed is marked anæsthesia found in the corresponding region. Thus, given a lesion of the cord at the level of the fifth dorsal vertebra, the seventh dorsal root will be injured at its emergence from the spinal canal, but not the fifth and sixth dorsal roots which at this point have an extramedullary situation; but as the sixth dorsal root helps to supply the region of the seventh and complete anæsthesia occurs only with lesion of the latter, the limit of anæsthesia in such a case will be found at the upper margin of the cutaneous region of the eighth root, that is, three vertebræ below the seat of the injury. If, however, the limit of the anæsthesia lies only the length of a single spinous process below the vertebral injury, we have to deal with the rare case of a simultaneous lesion of cord and root; then the vertebral injury is generally very serious.

At the cervical and lumbar cord these apparent discrepancies between vertebral lesions and limits of the anæsthesia are not so clearly manifest owing to the different and much more complicated arrangement of the functional regions of the several segments; still even there we must of course bear in mind that according to Sherrington's law (p. 579) a lesion which destroys, say, the seventh cervical segment, will produce marked sensory symptoms only in the region of the eighth.

The segmental diagnosis is altogether different and far more difficult when there are no evident deformities of the spinal column. But even in such a case it will be possible when the cord lesion is total in at least one segment, which is probably always the case in grave contusions. Then it becomes necessary to determine accurately the extent of the anæsthesia and of the muscular paralyses and, when the cervical or lumbar enlargement is involved, especially the atrophic muscular paralyses. Reference being made to the above table, it will be found which highest segment of the cord is to be considered as injured in the presence of such symptoms. Here again the rule must be followed that in general the lesion is to be located in the segment in question or in the corresponding roots at their emergence from the canal, but not in the roots in their extramedullary course in the canal; besides, the lesion must be situated in the highest segment implicated in the respective case. To recapitulate the facts in the inverse order, if in a concrete case we find a total anæsthesia in the cutaneous region of the eighth dorsal root, the seventh, according to Sherrington, must likewise be affected, and the seventh dorsal segment is opposite the fifth dorsal spinous process. In a similar manner the level of the segment may also be determined in cervical and lumbar lesions and,

as above stated, from the extent of the atrophic muscular paralyses we may deduce the inferior limit of the traumatic lesion proper.

In the absence of vertebral deformity and when the lesion of the cord is only partial, a segmental diagnosis of the lesion is impossible. In these cases, *e.g.*, in lesions high up in the dorsal cord, the anæsthesia may begin only in the legs or be entirely lacking. For the segmental diagnosis in the dorsal cord, however, we require especially the limit of the anæsthesia, since muscular paralyses with atrophy are not at our disposal. Again, in partial lesions in the cervical and lumbar enlargements which usually furnish us some guiding points as to their level, the important muscular paralyses may be so scattered and incomplete that the location of the injured segment cannot be recognized from them, at least not with certainty.

A few words may here be added about the prognostically important possibility of a differentiation of injuries of the conus medullaris and the cauda equina. When there is a distinct spinal deformity which is situated below the second lumbar vertebra, the cauda alone can be injured, as the cord does not extend so far. But otherwise a positive decision is usually very difficult, since in this region are crowded into a narrow space the several segments of the lumbar and sacral cord, as well as the extramedullary roots, and the sacral portion of the lumbar enlargement, for instance, is accompanied not only by its own but also by all the roots of the lumbar plexus. Ordinarily, in lesions of the cauda as well as those of the lower portion of the lumbar enlargement, the lumbar roots are not implicated and the functional disturbances are confined to the sacral plexus; in grave cases, however, all the roots of the cauda or of the sacral portion of the cord with the passing roots of the lumbar plexus may be destroyed, and in either case there is total paraplegia of the legs. Under such circumstances a positive differentiation between lesions of the cauda and conus is impossible. Of course the total paraplegia may also be due to a complete destruction of the entire lumbar enlargement. Evidently in caudal lesions there can be no combination of an atrophic paralysis in the lumbar plexus (*i.e.*, especially in the quadriceps muscle) with a spastic paralysis in the sacral plexus (*i.e.*, in the leg and foot); this would lead us to expect a loss of the patellar reflexes with clonus of the Achilles tendon. Such a grouping, therefore, will point positively to a lesion in the cord itself, though it will certainly be very rare, in traumatisms in particular. Besides, in caudal lesions the pains are of special severity and the paralyses as a rule are not so symmetrical as in destruction of the cord proper.

Is it occasionally possible for us to recognize early whether the

lesion is partial or total with reference to the cross section? We have seen above that a unilateral lesion only is the common result in stab-wounds. The lesion of the cross section is sure to be partial when with clearly recognizable deformity of the spinal column the extent of the functional disturbances does not correspond to the level of the lesion, but the sensory disturbances, for instance, remain far below it. It is sure to be total when with lesions in the dorsal and cervical cord the tendon reflexes remain absent beyond the time of a possible effect of shock, and the paralysis of the leg continues flaccid. In all other cases only the further course will decide this question.

In the preceding remarks the topographical relations of the several vertebræ or their spinous processes to the several segments and roots of the cord have been frequently spoken of. Vertebro-medullary topography forms a most important chapter; for when these relations are accurately known, we are able to localize a lesion of the cord even when no injury can be discovered in the spinal column. Such a diagnosis is demanded in the event of possible operative interference. Since we have to rely upon the dorsal spines in determining the level at the vertebral column, the relations of these processes to the several segments of the cord or to the sources of the roots are of paramount importance, as are in a lesser degree the relations of the spinous processes to the roots in their intervertebral course, because in severe cases the extramedullary roots may also be injured.

These relations are complicated by two conditions. First, by the more or less long intervertebral course of the roots from their origin in the cord to their emergence from the vertebral canal. Only in the uppermost cervical segments are the origin and emergence at the same level; farther down this relation changes more and more. At the dorsal cord the difference corresponds to about the space occupied by two vertebræ, and all the sacral nerves originate about opposite the first lumbar process, while they emerge only from the sacrum. These circumstances would render a local diagnosis very difficult were it not, as repeatedly stated above, that experience has shown that it is usually the cord itself and not the extramedullary roots which is destroyed by a traumatism, aside from special conditions which are easily recognized. But this fact being known, they explain at once that in most cases of dorsal-cord lesions the limit of the anæsthesia, even apart from Sherrington's law, must be located far below the level of the bone lesion.

Moreover, the tips of the spinous processes, which alone interest us in this connection, are about opposite the middle of their respec-

tive vertebral bodies only in the lumbar region. At the cervical portion they correspond about to the lower edge of their bodies, at the three upper dorsal vertebræ they correspond to the upper edge of the body below; the fourth to the eighth dorsal spinous processes extend farthest down and correspond about to the middle of the next vertebral body; the last four dorsal processes gradually approach again the lower edge of their own bodies.

The first three cervical processes are opposite the segmental region of the third, fourth, and fifth cervical nerves. The sixth cervical nerve originates about in the interspace between the fourth and fifth, the seventh nerve between the fifth and sixth cervical processes; the sixth process corresponds to the origin of the eighth cervical nerve, the seventh process to that of the first dorsal nerve. In the thoracic portion the processes correspond about to the roots bearing the next two higher numbers; thus, the third to the fifth, the fourth to the sixth, etc., the tenth to the twelfth pair. The first thoracic process corresponds to the interspace between the second and third dorsal root, the second to that between the third and fourth pair of roots. The roots of the lumbar and sacral plexus are all between the eleventh dorsal and the first lumbar spinous process.

The cervical enlargement, which ends at the first dorsal nerve inclusive, corresponds about to the spinous processes of the cervical vertebræ; the lumbar enlargement to the spinous processes of the last two dorsal and the first lumbar vertebræ.

It should be added that there may also be considerable individual variations in the vertebro-medullary topography. Thus, according to Reid, in some cases the process of the seventh cervical vertebra may reach the lower edge of the eighth cervical segment; in others, the upper edge of the third dorsal segment. In general, however, after a positive segmental diagnosis, it will be possible to locate the lesion correctly with reference to the corresponding vertebral processes by means of the schematic diagram above given.

Prognosis.

The prognosis of severe cord lesions is always very grave; in the great majority of cases death follows. Cases which terminate fatally at once in consequence of the injury, or soon afterwards of asphyxia or of a septic infection, do not of course enter into the prognostic consideration. Some guiding points as to the outcome have already been implied in the discussion of the course. In general it may be stated that the higher the seat of the lesion and the more complete its transverse extent, the more surely and rapidly will

death ensue; in how far these circumstances may be diagnosticated has been fully considered in the previous section. Serious injuries of the spinal column also render the prognosis more unfavorable, since they easily give rise to infection.

When convalescence and improvement occur, patients with lesions of the dorsal cord are better off than those with lumbar lesions, because a spastic paralysis does not prevent walking, while a flaccid paralysis does, and because the vesical disturbances are more severe in lesions of the lumbar cord.

Unilateral lesions furnish a favorable prognosis *quoad vitam* unless infection occurs. Luxations of the cervical vertebræ do so only when the cord has been but slightly injured and reduction has been prompt. When the cord is damaged, immediate death is almost invariable.

Treatment.

A direct causal therapy is really applicable only in luxations of the upper cervical vertebræ. If such a case has not been immediately fatal, we may assume that the injury of the cord is merely slight and after prompt reduction complete recovery may be hoped for. Great care, however, is necessary, for not rarely it is precisely during attempts at reduction that a fatal compression of the cord is effected, for instance, by the breaking off of the odontoid process of the axis or by this process when broken being forced into the cord. In cases complicated with luxation and fracture, especially at the thoracic portion of the spine, it is better to dispense with attempts at reduction, for they are almost never successful.

When asphyxia is present from the first or septic infection manifests itself early, any treatment is of course hopeless and our object must be to relieve pain when present, for which purpose morphine should not b espared.

In most of the remaining cases it will be important to avoid grave complications, especially bedsores and cystitis, and to preserve the patient's strength as much as possible. Transportation of the patient requires great care. His sacrum should rest on a water pillow; the heels and the region of the spines of the scapula which are also liable to bedsores may be protected by special cushions. If bedsores develop in spite of these precautions they should receive the best surgical treatment. A frequent change of position of the patient with a view to the prevention of decubitus is not advisable, since additional lesions of the cord may be caused thereby if the vertebral column is injured. Extreme cleanliness of the body is imperative, especially if the patient is liable to soil himself. If catheterization is required,

the most rigid asepsis must be maintained. The faeces, which are usually retained and very hard, had best be removed mechanically. The diet should be very nutritious and free from stimulating substances and those liable to cause flatulence. These simple hygienic measures will suffice, in those cases in which it is at all possible, to give the cord time to recuperate and to bring about a partial recovery when the lesion is not total, or even complete restoration to health in simple compression from hemorrhage. In most cases, however, the fatal issue is simply delayed; ultimately bedsores develop in spite of our efforts, and more often still cystitis, pyelitis and nephritis, or general marasmus follows, and the patient finally succumbs to a hypostatic pneumonia.

Owing to this hopelessness of the treatment in many cases, the attempt has been made to improve the prognosis of the affection by operative interference. On the whole, however, the results have been but moderately successful, as might have been expected. Optimistic views as to operative measures can prevail only when the relations are imperfectly known. Let us consider what is to be effected by an operation. We might aim at the removal of effused blood from the meninges. But this is usually resorbed spontaneously, and hence so serious a measure as a trepanation of the spinal column is hardly justifiable. Or else we may have in view a reduction of a grave dislocation which causes pressure upon the cord. Often, however, this is not feasible even after trepanation, and the idea of making room for the compressed cord by a laminectomy is erroneous because the pressure on the cord is not caused by the arch but usually by the body of the vertebra. The removal of a splinter of bone pressing upon the cord would be rational, but we have seen above that we can never positively recognize a permanent compression of the cord by the spinal column after an injury, nor can we exclude it with certainty. Hence the operation is always a step in the dark, and even if we were fortunate enough in such a case to find a splinter of bone compressing the cord—on the whole a rare occurrence—the cord is apt to be so seriously damaged that a restoration of its function is not to be expected.

As a rule, therefore, operation is to be rejected in cord lesions. But there are two exceptions. Chipault has demonstrated that in cases of fracture of an arch with slight injury of the posterior portions of the cord, the removal of the fractured arch may be of decided advantage. This is very plausible, and when such a fractured arch is found its removal is to be advised. Moreover, injuries of the cauda equina present, according to Thorburn and Chipault, a favorable opportunity for operative interference. The fibres of the cauda

equina behave like peripheral nerves; even after serious and long-continued compression they may, when this is removed, become restored by an outgrowth into the periphery of the central axis cylinders of the compressed nerves. Chipault advises, in the presence of marked deformity below the second lumbar vertebra in which the cauda equina alone can be implicated, to operate at once unless material improvement has already occurred. In the latter cases, however, as we have seen, the differential diagnosis between injuries of the cauda and those of the conus is often impossible.

At any rate we must abstain from any operation when the symptoms indicate a total contusion of the cord. We know that this is to be diagnosed when in cases of dorsal or cervical lesion with complete anæsthesia the tendon reflexes remain permanently absent and the paraplegia continues flaccid.

When convalescence sets in and progresses to some extent, a treatment at some indifferent or thermal-saline spa, such as Teplitz, Wildbad, or Nauheim, will be suitable, together with massage, gymnastics, and electricity.

LESIONS OF THE CORD FROM DISEASES OF THE VERTEBRÆ.

Spinal Caries.

History.

We are indebted for our first knowledge of the different forms of paralysis occurring in caries of the spine, and dependent on an involvement of the spinal cord, to Percival Pott, an English physician, who first described the affection in 1779. For a long time the opinion was unreservedly entertained that the spinal-cord symptoms in caries of the spine were produced by a compression of the cord from the bones involved, although clinical experience demonstrated very early that great deformity of the spine may occur with absence of any lesion of the cord, that the symptoms of paralysis may disappear in Pott's disease when no change has taken place in the deformity, and thirdly that they may occur in cases in which no deformity has at any time been present. When, therefore, a more careful histological examination demonstrated later on that the extradural tissue and the dura mater itself were usually involved in the tuberculous process, and when furthermore there were found distinct changes in the spinal cord itself which were looked upon as being of an inflammatory nature, the inflammation theory took the place of the compression

theory; a myelitis of the cord was spoken of in these cases—a compression myelitis. This theory was upheld especially by Charcot and Michaud, and found adherents also in Germany. It was not, however, accepted in its very beginning by all, and Kahler demonstrated that the histopathological changes which were looked upon as the result of inflammation might occur simply as the result of a collateral œdema from slight compression. Strümpell disbelieves in the existence of a compression myelitis, referring everything to the result of mechanical compression, caused, not by displaced bone, but by extradural fungosities. Erb takes a middle position. Latterly, the work of Schmaus, to which we shall have occasion to refer frequently in this article, has had a marked influence on the conception of the pathogenesis of Pott's paralysis.

Pathological Anatomy.

Caries of the spine consists in a tuberculous disease of the bones of the vertebral column. In by far the greater number of cases the seat of the disease is in the vertebræ, generally in the spongy substance of the bodies; much more rarely the disease originates in the small articulations of the spinal column, and still more rarely the vertebral arches are diseased. Macewen thinks that the disease may occasionally remain behind in the arches after the more severe affection of the bodies has disappeared, and that the lesion of the cord may be maintained from this point. In the cases mentioned by Macewen, we have probably not so much to do with a still active disease of the bones as with fungosities on the posterior surface of the dura, which have developed into cicatricial tissue, still capable of exerting pressure.

If the caries should confine itself to the body of a vertebra, and perhaps even to the centre of the latter, the process may go on to healing without having at any time endangered the cord itself. As a general rule, however, we do not meet with such a limitation of the process. The latter extends through the intervertebral cartilages to the next higher or lower vertebra, and also extends through the posterior bony layer of the body, first causing a projection forwards of the periosteum towards the dura, and finally perforating the former and invading the extradural tissue, which consists mainly of fat. In this situation a more or less extensive fungoid growth, containing cheesy and purulent foci, will now be developed and will press against the anterior surface of the spinal cord. Should the opening in the bone be only a small one, the fungous growth may at first be attached to the bone with a pedicle, like a mushroom, but afterwards it ex-

tion cease and the œdema disappear, the arrested function may be again restored. This œdema may, as stands to reason, also make its appearance rapidly, and even in a spinal cord which has up to that time been in a perfectly normal condition; in the same manner it may rapidly extend from a circumscribed diseased region. These cases especially have given rise to the supposition that acute myelitis makes its appearance in caries of the spine as a sequel to the more chronic spinal-cord affection. The spinal cord does not of course become narrowed in these cases, but may retain its normal volume, or even become swollen. Its consistence remains soft. When the œdema persists, definite anatomical lesions may result. The swelling and destruction will involve more and more nerve fibres, the ganglion cells even not escaping; the place of the destroyed nerve tissue is taken by granulation cells, the blood-vessels increase in number, and small-celled infiltration of the connective tissue may even take place; finally the whole of the cord substance becomes softened and escapes readily on section, a small ribbon of nerve substance immediately below the pia alone remaining intact. If now the softening process does not extend to other regions, the disintegrated cord substance may gradually become absorbed, the remaining portion, which usually consists of fungating glia and normal connective tissue, comes in contact. In the enormously shrunken cord substance, which is sometimes flat as a ribbon, we can hardly ever find even remnants of nerve tissue on section. Naturally the final destruction may in this case involve only a portion of the cross section, and a partial recovery of function may result from the disappearance of the œdema in the other parts.

In short, we have to deal in by far the greater number of cases of disease of the spinal cord accompanying caries of the vertebræ with either a simple compression or œdema, and when this condition has been of long duration, with destruction, softening, and cicatricial formation. A true myelitis, however, is not present in these cases, and what has been regarded as such, clinically and anatomically, has usually been the result of a more or less rapid œdema or of compression.

Ischæmic or anæmic softening from the occlusion of blood-vessels of the cord, in spinal caries, is looked upon by Schmaus as of very rare occurrence; it may, however, under certain circumstances occur. Furthermore, according to Schmaus, the œdema is not always necessarily a simple one, caused mechanically, but there may also occur an inflammatory œdema containing toxins which may themselves occasion a true myelitis. This occurrence is, however, up to the present a purely hypothetical one.

As has been mentioned above, it is only in very rare cases of ver-

tebral caries that perforation of the dura with extension of the fungoid inflammation to the pia occurs. If it does take place, a true myelitis may follow, but it will then be of a tuberculous form. Raymond indeed regards this as a not very infrequent occurrence. Oppenheim in one such case has also observed a disseminated tuberculous myelitis. We need only mention that rare cases of tuberculous pachymeningitis externa have been observed in which the bone itself was not involved. The effects on the cord and the clinical symptoms may in this case be the same as in caries of the spine, but deformity will be absent.

In severe lesions of the cord, ascending and descending forms of degeneration will of course make their appearance. The spinal nerve roots which pass through the region of the fungoid inflammation are subject to the same injuries as the cord, although they are more resistant than these. They may thus, for example, be simply compressed as they emerge from the spinal canal, or may become œdematous. In either case, if the bone affection progresses, they become degenerated or disintegrated. A direct inflammatory process may occur much more readily in the nerve roots than in the cord. The muscles which are dependent on the diseased portion of the cord or on the diseased anterior roots will then become degenerated and atrophied.

Etiology.

Caries of the spinal column occurs in both sexes with equal frequency. It is as a general rule a disease of early life and especially of childhood. It rarely makes its appearance before the fourth year of life, but occurs quite frequently about the time of puberty. However, it does not respect even old age, and the first case which the writer (Bruns) had the opportunity to observe closely was that of a woman about sixty years of age; he has met with a number of cases between thirty and forty years of age. Gowers indeed holds that tuberculosis of the spinal column is the most frequent form of tuberculosis occurring during the second half of life.

Caries of the spinal column is a tuberculous process, and we often find in these patients other tuberculous foci, especially in the lungs or in other bones and joints and in the lymphatic glands. Occasionally caries of the spinal column occurs as an isolated lesion. A general miliary tuberculosis makes its appearance late, if at all, after the disease has existed in the bones for some time.

Frequently a traumatism, especially one of the spinal column, is asserted as the cause of caries, and it is certain that the latter is often brought to light as a sequel of such injury. Either latent

tuberculous foci were already present in these vertebral bodies, and were incited to more rapid growth by the injury, or else the tubercle bacilli, probably already present in the organism, found in the injured parts favorable conditions for growth. In either case the traumatism is therefore only a remote cause of the spinal caries.

Symptoms.

The symptoms of Pott's disease may be divided into those referable to the diseased vertebræ, to the affected nerve roots of the spinal cord, and to the spinal cord itself. As a general rule they also follow this order in their appearance. It may be stated in advance that fever may also be present in all the stages of tuberculous caries of the spine, as in all other tuberculous affections. Its degree may vary greatly, and it may be absent altogether.

Disease of the vertebræ makes itself known in the first instance by pain. The pain is not always correctly located by the patient, and it is then necessary to trace it to its origin. Pressure should be made with the finger tips on the spinous processes, or better still an attempt may be made to displace these laterally; this usually causes excessive pain. The seat of the disease may also reveal itself by great tenderness when a hot sponge is passed over the spinal column. The method of eliciting pain by violently striking the skull is rather rough, and besides does not always clear up the question as to which vertebræ are involved, as the patient is seldom able to localize the pain produced in this manner. As we meet with tender vertebræ also in hysteria and neurasthenia we must especially endeavor to exclude these diseases. As a general rule the pain in these diseases is not so localized, and it is also not so dull, deep-seated, and persistent; its seat is especially in the skin, and it is not increased by firm pressure. The pain is of course increased by motion. The patient seeks to avoid this and therefore holds his spinal column as rigid as possible. This stiffness or contracture constitutes the second symptom of vertebral disease. The symptom is most marked in the cervical portion of the spinal column, which has the greatest mobility in health. Here the patient supports his head as much as possible with his hands, when lying down or rising, in order to prevent even the slightest movement of the vertebræ. Not infrequently the head is also carried somewhat flexed and turned to one side so as to remind one of torticollis. If the seat of the caries is in the dorsal region of the cord, the patient, when attempting to pick up something from the floor, does not incline the trunk forwards, but bends his knees, at the same time holding the trunk perfectly straight. Cough-

ing and sneezing, as well as all jarring of the body, may increase the pain greatly.

The most characteristic symptom finally is the deformity of the spinal column, the acute angular kyphos or gibbus. It is chiefly met with in the dorsal region of the spine. Rachitis and other diseases of the vertebræ, excepting traumatisms, do not produce it. It is formed by the collapse of the body of one of the vertebræ, so that the spine above inclines forwards and the spinous process which lies above or below the diseased part, usually the lower one, strongly protrudes. The protrusion is most marked if only one vertebra is destroyed; when more are involved the kyphos is more rounded. The spinal column may be occasionally deflected to one side. It must, however, be specially observed that kyphosis frequently occurs only very late, after the lesion of the spinal cord has already reached its full height, and that it may be absent during the whole course of the disease. The cold abscesses and other neighboring affections which may be present as complications have already been discussed.

The nerve-root symptoms which may accompany caries of the spinal column may be dependent on lesions of the posterior as well as of the anterior roots, and are therefore sensory and motor in character. In both regions we have accordingly to distinguish between irritative conditions and conditions of paralysis—that is to say, between pain or hyperæsthesia and anæsthesia in the sensory field, and between spasms and generally flaccid paralysis in the motor. As a rule the sensory symptoms predominate over the motor, and in the former the symptoms of irritation again predominate over those of paralysis. Pain or hyperæsthesia is usually present in certain skin areas, but a marked anæsthesia, which may be referred wholly to the lesion of the vertebræ, is much more rare. Spasms have indeed been observed in pure root lesions but they are very rare. The paralysis and atrophy due to an affection of the anterior roots appear as a rule only late and very slowly and gradually, taking a pretty long time before they become fully developed in a certain muscle. It is doubtless because of this slow course that we are only rarely able to demonstrate a complete reaction of degeneration in the affected muscles; generally we have to deal with only quantitative changes of the electrical excitability—that is to say, a lowering of the latter which gradually becomes lost. We can understand this from what we know at the present time of the extensive anastomoses of the spinal-cord roots in the plexuses and in the peripheral nerves—anastomoses which lead to the supply of each muscle and skin area by at least three roots; and of the demonstrated fact that complete anæsthesia of

any skin area and marked paralysis and atrophy of a definite region can obtain only when all the posterior and anterior roots supplying the part are injured. For the occurrence of pain or hyperæsthesia in a certain skin area or for the appearance of spastic symptoms in certain muscles, the irritation of one of the roots involved is quite sufficient; but for the production of anæsthesia, or of paralysis and atrophy all must be markedly injured. The sensory and motor root symptoms are confined in their localization to the diffusion of the individual roots of the spinal cord, they will not therefore correspond to the area of diffusion of the peripheral nerves. We cannot stop to study this more in detail here, but what is most necessary has already been said in the chapter on injuries to the spinal cord.

The pain is generally tearing, lancinating, or it may be a violent burning sensation; the anæsthesia involves all the forms of sensation equally.

In rare cases trophic disturbances of the skin belonging to that area are induced by the affection of the nerve roots of the spinal cord. Most frequently herpes zoster is present, but a variety of pemphigus has also been observed.

In a special sense, the grouping and localization of the root symptoms is of course dependent on the seat of the caries as regards the longitudinal axis of the spinal cord. In the most frequently occurring site of caries in the dorsal region of the spinal column marked motor symptoms depending on root lesions are not prominent, and only when the seat of the disease is pretty deep a paralysis of the abdominal muscles might occur. The girdle pain involves one-half or the entire circumference of the thorax at any height; it does not confine itself to an intercostal space but takes its course according to the root areas, running horizontally around the chest, thus crossing a number of intercostal spaces. If anæsthesia and herpes zoster are both present they will be found in the same area. When a gibbus is present, the pain is always felt, most markedly at least, two spinous processes below the former. If the upper dorsal and lower (seventh and eighth) cervical nerve roots are affected by the caries, the pain, possibly anæsthesia, and later herpes, will be situated on the ulnar side of the arm and forearm and probably the hand; the flaccid atrophic paralysis involves the small muscles of the hand and the extensors and flexors of the fingers and hand. Painful tonic spasms in these muscles have also been observed in cases of lesion of the anterior roots. An affection of the first dorsal root causes a contraction of the pupil and a narrowing of the palpebral fissure, which are especially noticeable when unilateral. If the upper roots of the cervical enlargement are affected, the seat of the pain will be in the shoulder

and arm; any anæsthesia present will involve the radial side of the arm. Paralysis and atrophy will be found in the deltoid muscle, biceps, brachialis internus, supinator longus, infraspinatus, and perhaps in some other muscles of this region. The root symptoms in tuberculous disease of the upper cervical vertebræ, which is not very rare, are quite characteristic. The pain is here experienced in both occipital nerves. A unilateral atrophy of the tongue has occasionally been observed in these cases by the writer and others. In the same manner the accessory nerve may also be injured and a paralysis of the sternocleidomastoid and the trapezius may occur. The writer (Bruns) once observed in a case of displacement of the upper cervical vertebræ, in addition to a paraplegia, an aphonia so complete that it could not be explained wholly by the paralysis of the muscles of respiration. As a rule, the small muscles, which are concerned in the movements of the head, are also paralyzed.

When the seat of the lesion is opposite to the upper portion of the lumbar enlargement, the root symptoms, pain, perhaps anæsthesia, and possibly zoster, if present, have their seat on the anterior surface of the legs; the paralysis and atrophy involve the crural and obturator muscles and those supplied by the peroneal nerve. Should the lesion be situated in the lower portion of the lumbar enlargement, the pain will be felt in the ischiatic region, over the posterior surface of the legs, in the perineum, and in the genitals; anæsthesia and atrophic paralysis of the muscles of the calf, of the crural flexors, and of the muscular tissue of the perineum may also occur. In lesions of the cauda, the sacral roots only are usually affected, but the lumbar muscles may also be involved.

To recapitulate: The root symptoms, especially the most common one, pain, may be of variable intensity and duration in cases of spinal caries. They are on the average of moderate intensity, more moderate than in other diseases of the vertebræ, especially than in carcinoma. They may be, however, of quite extraordinary intensity or they may be absent during the whole course of the disease. As a rule they do not exist alone for any length of time without marked bone or spinal-cord symptoms. They usually follow the signs of disease of the bones, and precede the cord symptoms; not very infrequently they are not seen until after marked symptoms of compression of the cord have appeared. They are nearly always bilateral. As a general rule they are more frequent, more violent, and more disseminated in disease of the lumbar and sacral roots, and especially of the cauda equina, than in caries of the spinal column of the thorax. Caries of the cervical vertebræ is also more painful than that of the dorsal spine. The symptoms which occur from the involvement of

the cord itself in vertebral caries are dependent on the functional disturbance of those parts which are directly attacked by the lesion, and on the interruption of communication in the parts of the spinal cord which lie below the site of the lesion. As the functions of the single segments do not differ from those of the roots which lead from them, the symptoms due to lesion of the cord itself at a point corresponding to the seat of the caries do not materially differ from those due to injury of the nerve roots emerging at the same level, especially if the roots are involved just as they arise from the cord, as is usually the case. We therefore have to deal in the motor area with a flaccid paralysis accompanied by atrophy and loss of the tendon reflex, in the sensory area with hyperæsthesia or anæsthesia. If the root symptoms have been very pronounced before the cord itself has become involved, the subsequent implication of the cord will not be marked by the occurrence of any new symptoms in the area dependent on the affected segment. As a general rule, however, these symptoms will not be of so high a degree in a lesion of the roots, and we may, therefore, since the roots are generally more resistant to injury than the cord, refer the occurrence of marked paralysis and atrophy and distinct anæsthesia rather to a lesion of the cord than to one of the roots. This applies, however, only in a general way, and in special cases we shall meet with all kinds of irregularities. If both root lesions and segment lesions are present, it will hardly be possible to refer the individual symptoms to one or the other, although violent pain is probably always due to irritation of the root. As the segment symptoms are produced by the direct injury of the cord at the site of the fungoid growth, they are located higher up than the root symptoms, and we may conclude from them as to the height of the lesion and its upward extension; always, of course, provided there is not some functional disturbance of the cord above the site of the compression. The grouping and localization of the functional disturbances due to the segment lesion of the cord are the same as those caused by the lesion of the corresponding roots, which we have described above.

The functional disturbances of those areas which are dependent on the portion of the cord lying below the lesion are due to the more or less complete interruption of the conduction of the cord by this lesion. They are in their way always the same, only differing according to the height of the compression. They are the only symptoms which justify the conclusion that the cord is itself involved in the disease process, for, as regards the symptoms referable to the site of the compression, we frequently are unable, as before mentioned, to decide whether we are dealing with cord or with root symptoms.

When, however, the roots alone are involved, there is no interruption of conduction in the cord. The symptoms of interruption of cord conduction consist in disturbances of motility, of sensibility, of the reflexes, and of the sphincters. The paralysis is not combined with atrophy and disturbances of electrotonus, and it is nearly always spastic, both of which characters are in contrast to the paralysis which is directly produced at the site of the compression. Sensibility is usually equally disturbed in all its forms, if it is affected at all. The skin, and especially the tendon reflexes, are increased if their reflex curve lies below the site of compression. The disturbance of the sphincters varies according to the site of the compression, and it is impracticable to make any generalization concerning them here.

Let us assume that the lesion of the cord has led to an incomplete interruption of conduction in the compressed segment, we shall then meet with the following group of symptoms, according as one or the other part of the cord is affected. We will first mention those symptoms which are referable to the segments which are directly injured, or the corresponding nerve roots, and then the symptoms due to the interruption of conduction in the cord.

Dorsal Region.—Girdle pains, a zone of hyperæsthesia at a level corresponding to the site of the lesion. Spastic paralysis of the lower extremities accompanied by contracture of the extensors, rarely of the flexors, and by clonus of the patellar and Achilles tendons and frequently a marked so-called "spinal epilepsy." Anæsthesia as far as the skin area of the directly compressed segment, but increased skin reflex. Difficulty of micturition frequently accompanied by tenesmus and sometimes going on to total retention. Frequently ischuria paradoxa, rarely true incontinence of urine; sometimes very frequent alvine discharges, at other times constipation.

Cervical Enlargement.—When the lesion involves the lower half of the cervical enlargement, there will be pain or hyperæsthesia of the ulnar surface of the arm and forearm and of the hand. Atrophic paralysis of the small muscles of the hand and sometimes of the flexors and extensors of the fingers and hand, accompanied by reaction of degeneration. Anæsthesia of the thorax reaching anteriorly as far as the second rib and posteriorly as far as the spine of the scapula, and involving the ulnar half in the arm. Contraction of the pupil and narrowing of the palpebral fissure. The conditions of the paralysis in the lower extremities, of the tendon reflexes in this situation, and of the disturbance of the sphincters, are the same as when the seat of the lesion is the dorsal region of the cord. In addition, however, the muscles of the chest are also paralyzed and respiration is difficult.

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When the lesion involves only the upper half of the cervical enlargement, there may be spastic degenerative paralysis in the forearm and hand, and atrophic degenerative paralysis in the shoulder and arm. The pain will be seated in the shoulders and arms. The anæsthesia involves the arms. The pupils and palpebral fissures are not contracted (?). The other conditions are the same as those mentioned in the preceding paragraph.

When the lesion attacks the *upper cervical vertebræ*, a spastic paralysis of all four extremities will exist. Anæsthesia will also involve the nucha, the upper portion of the chest, and the neck. The diaphragm is usually paralyzed. At times the medulla oblongata is also involved, and Charcot has called attention to the slow pulse accompanied by attacks of syncope in some of these cases. Epileptic seizures have also been observed.

In lesions of the *lower dorsal vertebræ* and of the *upper lumbar vertebræ* the whole lumbar enlargement may be involved. We then find flaccid, atrophic degenerative paralysis of the lower extremities, and the tendon reflexes are absent; there is incontinence of urine, although the bladder may be able, on account of the elasticity of the sphincters, to retain small quantities of urine. Impotence is also present. Both lower extremities are destitute of sensation, and pain, if present, is located either in the abdomen or in the inguinal region, or possibly on the anterior surface of the thigh. Should the lumbar cord proper be involved, we may find flaccid atrophic paralysis with absence of the patellar reflex; in the leg there will be spastic paralysis accompanied by clonus of the Achilles tendon. The anæsthesia will extend as far as in involvement of the whole lumbar enlargement. Pain is also experienced in the crural region. The same functional disturbances of the bladder may exist as are found accompanying lesions of the dorsal region of the cord.

When the *sacral cord* only is involved, atrophic degenerative paralysis is present only in the muscles of the legs and of the feet, and sometimes in the flexors of the thigh; the remaining muscular tissue of the thigh may be unaffected, the patellar reflex may be preserved, but the reflex of the Achilles tendon is absent. The anæsthesia involves especially the posterior surface of the leg and thigh, the genital organs, and the perineum. The disturbance of the sphincters is the same as when there is a total lesion of the lumbar enlargement.

If the *cauda equina* is affected by the lesion, the symptoms are generally the same as when the sacral portion of the lumbar enlargement is affected. As a general rule the lesion in caries of the spinal column is not so exactly limited, and the partial affections of the cervical and lumbar enlargements are for this reason probably rarely

met with. In the lumbar region of the cord a gradual lesion of the sacral portion of the lumbar enlargement, without an affection at the same time of the lumbar roots leading to the cauda equina, will be very rarely met with; it is, however, possible. Decubitus of the sacrum may occur in all cases in which marked anæsthesia is present; cystitis in all cases in which marked disturbance of the bladder is present.

The symptoms described so far are those which occur when the lesion has caused a severe but not total interruption of conduction. They are changed as well when the interruption of conduction is still more incomplete as when it is a total one. In the first instance the spastic paralysis becomes a spastic paresis, and the atrophic paresis at the site of pressure will itself be very slight. The anæsthesia becomes a hypæsthesia, and in a great number of cases there is no disturbance whatever, even when the motor paralysis is quite marked. In the same manner the disturbance of the sphincters may be rudimentary. When the lesion involves the dorsal region, its frequent seat, there may be present as the only cord symptom a spastic paresis of the lower extremities; should this paresis advance to a paralysis, sensory and sphincter disturbances will also appear. When such slight lesions affect the lumbar region of the cord a flaccid paresis will manifest itself, and the trophic disturbances will also be slight.

When the interruption of conduction is total, the tendon reflexes of the lower extremities will also be abolished, even in lesions of the cervical and upper region of the dorsal cord. The paralysis will then be of the flaccid variety. This condition may follow a spastic paralysis in association with a gradual increase of the interruption of conduction. The sphincters are then probably affected in the same manner as in lesions of the lumbar region of the cord. Babinski has described cases in which the patellar reflex was absent, even in very slight lesions of the region of the dorsal cord; this circumstance is difficult to explain, but such cases are interesting in a therapeutical relation.

The cord symptoms in caries of the spine are almost without exception bilateral, even when they are not altogether symmetrical. Occasionally, however, only one side is affected, and then paralysis may occur on the side of the lesion, and anæsthesia on the opposite side as in Brown-Séquard's paralysis. The writer has seen this very distinctly in a case of caries of the cervical vertebræ.

Course, Duration, Termination.

Vertebral caries and the spinal paralysis occasioned by it constitute, as a rule, a chronic disease. In typical cases, as already stated, bone, root, and cord symptoms follow each other, although a great many exceptions to this typical sequence occur. If the deformity of the spinal column is the primary symptom, paralysis usually follows it at an interval of some months. Cases have been known, however, in which the spinal symptoms did not make their appearance until years after the deformity occurred, and some even are on record in which kyphosis appeared in childhood but the paralysis did not occur until adult life. According to our experience true spinal symptoms make their appearance very late, if at all, in caries of the upper cervical vertebræ, for the reason probably that the severity of the symptoms associated with caries of this region usually insures an early resort to treatment.

In other cases the bone symptoms, especially the deformity, and the root and cord symptoms make their appearance at about the same time, and as a rule the whole process takes a more rapid course in these cases. In others again, which cases are not so very infrequent, the first symptoms are those referable to involvement of the nerve roots and cord, or perhaps to the cord alone, and then a total paralysis occurs without the slightest warning in the shape of symptoms of disease of the vertebræ. The deformity in these cases frequently follows a long time afterwards, or may never occur. It is not often that root symptoms, especially pain, exist for a long time as the sole evidence of disease. Of the greatest rarity are the cases in which, after the existence for some time of bone symptoms, particularly of pain or perhaps of deformity, with a total absence of cord symptoms, a complete paraplegia makes its appearance suddenly, perhaps as the result of a very slight traumatism.

If, however, as is usually the case, the cord symptoms make their appearance slowly and gradually, they will occupy some time in arriving at their full height. Oppenheim says that the time is about one year. There will be present in such a case, for example in caries of the dorsal region of the spine, at first perhaps a slight girdle sensation, followed by spastic paresis of the lower extremity and exaggerated reflexes; gradually disturbances of sensation and of the bladder will appear, the paresis progresses to paralysis, the tendon reflexes may disappear, and bedsores are produced. Although the symptoms are usually bilateral, we also meet with cases in which at first only one leg is paralyzed, or in which symptoms of Brown-

Séquard's paralysis appear. In other cases the course may be a rapid one, even in the absence of acute compression, or periods of seeming cessation of the process may alternate with periods of exacerbation. In these cases we have in all probability to deal with a rapidly occurring and again disappearing œdema.

A cessation of the cord disease may occur at any time. It is characteristic that this favorable circumstance does not stand in any positive relation to the course of the bone affection. Thus, while the bone disease remains, the affection of the cord may improve in consequence of the discharge of an abscess externally or of the removal of pressure in some other way; it may, however, continue and progress after the bone affection is fully cured. Acute exacerbations may occur after a long period of quiescence, especially as a result of a slight traumatism; in a case mentioned by Gowers a violent sneeze was sufficient to start the process again.

The further course and the final termination of the cord affection may also vary greatly. In one series of cases a total cure of the paralysis occurs, and it is quite noteworthy that this recovery may take place even where the gravest symptoms have existed for years. The writer has seen recovery ensue in a case which had progressed to marked contractures of the flexor muscles and severe decubitus. Anatomically these cases are probably due to simple compression or to an existing œdema. Occasionally the evacuation of a cold abscess exerts a favorable influence. The affection of the vertebræ then heals, leaving behind it usually a marked kyphosis. Of the cord symptoms the first which usually improve are the sensory, then the bladder disturbances; but slight, usually spastic forms of paresis remain for a long time. Relapses also occur in these cases, although, as Gowers remarks, more rarely than we might expect from the nature of the disease. Gowers mentions a case in which there was complete recovery, interrupted, however, by numerous relapses. This same author makes a remarkable statement to the effect that he has seen spastic paresis coming on in the case of one who had recovered from spinal caries, unaccompanied by paralysis, in early life. The recovery is more frequently only partial. Most usually we have to deal in that case with a spastic paresis of the lower extremities accompanied by slight bladder disturbance, both of which conditions frequently remain permanently.

It is very rare, however, that there is no abatement in the severity of the symptoms. This may be explained by the fact that in the incurable cases, when very pronounced paralysis and anæsthesia are present, death usually results from decubitus, cystitis, nephritis, amyloid degenerations, or possibly tuberculous disease in other parts.

As a rule, a fatal termination is more common in those cases in which grave cord symptoms are present, but the time of its occurrence is, of course, very indefinite. It results more frequently from tuberculous disease of other organs than it does directly from the spinal caries and its cord complications.

Diagnosis.

The diagnosis of Pott's disease in characteristic cases is not difficult. It is as a general rule especially easy when the course is perfectly typical, when the deformity and the root and cord symptoms make their appearance in characteristic sequence, and also in those cases of paraplegia due to disease of the spinal cord in which at least a certain tenderness on pressure over the spinal column can be demonstrated.

In older persons in whom, as already mentioned, caries of the spine is by no means a rare occurrence, a differential diagnosis between a tumor of the spine, especially a carcinoma, or perhaps an aneurysm, will have to be made. Deformity of the spine and root and cord symptoms are present in both cases. The presence of other tuberculous foci is in favor of caries; in favor of carcinoma is the history of a previous removal, perhaps many years before, of a malignant growth of the breast or more rarely of the stomach. The deformity of the spine in carcinoma hardly ever takes the form of a gibbus; usually we meet with a great number of diseased vertebræ, frequently nearly all are involved, the whole spinal column sinks and the patient's height is actually reduced, but a sharp hump is not present. The symptoms of involvement of the nerve roots and of the cord in cases of caries may be very similar to those seen in cancer of the spine; in each case they make their appearance usually on both sides simultaneously; in carcinoma, however, the root symptoms as a rule seem to be much more violent, and probably exist frequently for a longer time alone than in caries. The writer has under observation at the present time a case of vertebral carcinoma in which a slow atrophic paralysis of the left arm, accompanied by violent pain and an eruption of herpes zoster has been developing for one and a half years, without a lesion of the cord being demonstrable. A long-continued bilateral sciatica should always make us suspect the presence of carcinoma of the spine. In my experience the pressure symptoms in cases of carcinoma of the vertebræ appear frequently very late, and only in a moderate degree, although a sudden paralysis accompanied by displacement of the vertebræ may of course occur, an instance of which the writer saw only a short time ago.

The diagnosis is not always so simple, however. Thus Schlesinger observed a compression paralysis of the spinal cord in a case of mammary carcinoma. The diagnosis was naturally cancer of the vertebræ, but a post-mortem examination disclosed caries. Carcinoma of the vertebræ is of course an absolutely incurable disease.

The case becomes much more difficult in the absence of any deformity, and when a paraplegia, accompanied by more or less violent pain, gradually occurs. If this should occur in a young person, we must be obliged to consider first the possibility of a caries of the spine; this supposition becomes nearly positive when exacerbations of fever and signs of tuberculosis of other organs are present, and especially when there is a distinct, circumscribed tenderness of the spinal column. When, however, all these differential points are absent, the diagnosis between caries and other diseases of the spinal cord may for some time, and occasionally always, remain in doubt. Thus a tumor of the meninges of the spinal cord, as well as a caries, might lead to a paralysis with violent pain, and tenderness of the spine might at the same time be present. The fact that in these tumors, in typical cases, marked and for a long time isolated root symptoms, which are nearly always unilateral at the beginning, are the first signs, and that cord symptoms, and these also very frequently unilateral and of the Brown-Séquard type, follow, may serve for a differentiation; pains in the bones are usually slight and are the last symptom to appear, while they are as a rule the first symptom in caries. The presence of a tumor would prove decisive. A tumor (not a cold abscess) of the spine cannot be cured without an operation.

Caries of the cervical portion of the spine is with difficulty and frequently not at all to be distinguished from a pachymeningitis cervicalis hypertrophica, at least in those cases in which the deformity is absent. Root and cord symptoms may be identical in both cases, so also may be the course and duration of the disease; recovery may also take place in both cases. Pachymeningitis cervicalis hypertrophica, however, is a very rare disease, and may furthermore follow a pachymeningitis interna or a leptomeningitis without disease of the bone.

Of the true affections of the spinal cord intramedullary tumors are the most important as regards diagnosis. In these cases, however, the pain is usually at first slight, and bone symptoms may be absent altogether. The pain is slight in syringomyelia, and this affection can also generally be distinguished by its characteristic group of symptoms. The accompanying bone affection and kyphosis will seldom give rise to a confusion with caries, although the writer has

seen this error made in one case. Mistaking caries for a progressive spinal muscular atrophy is scarcely possible.

In the absence of any indication of bone disease, and in the presence of slight pain only, we may occasionally be unable to avoid making a provisional diagnosis of chronic myelitis. We need only bear in mind, however, that most of the cases so designated are really cases of compression paralysis; whether they are caused by caries must then be determined by other considerations. In such cases a positive diagnosis may possibly be made by means of the Roentgen rays.

In neurasthenic subjects, pain and tenderness to pressure in the spinal column together with subjective weakness of the lower extremities and increased reflexes, may lead us in the beginning to suspect caries; it would be more unfortunate if the reverse happened and caries were to be mistaken for neurasthenia. Oppenheim gives as diagnostic points in such cases the rigidity of the spinal column and the fact that in caries the arms are usually not affected. Undoubtedly, hysteria may also simulate caries of the spine. Occasionally this is done in a surprisingly realistic manner. The writer has seen a girl, thirteen years old, who had deformity in the region of the cervical spine accompanied by pain, a flaccid paralysis of the right arm, and spastic weakness in both legs, which came on gradually. The tendon reflexes, however, were not increased in the lower extremities, there was a total anæsthesia of the right arm within the boundaries characteristic of hysteria, and the paralyzed arm did not fall as lifeless when raised and then dropped, as would have been the case in one organically paralyzed. A cold douche and a single application of electricity brought about a cure at once. This case furnishes us with points in the differential diagnosis between hysteria and caries, which we need not repeat here. Moreover hysterical symptoms, especially marked anæsthesia, are not infrequently combined with those of caries, and what is of greater importance, there may occur in later life after the caries has healed and more or less perfect recovery from the paralysis has taken place, hysterical attacks which may simulate a relapse of the Pott's disease. The diagnosis is then of course frequently very difficult.

A knowledge of the exact location of the cord lesion is necessary for a complete diagnosis, for we should not be content with the general one of "caries of the spine and paralysis." This is of course easy, when marked bone symptoms are present. When these are absent the regional diagnosis must be made according to the extent and grouping of the symptoms, and our knowledge of the function of the individual segments. The segment diagnosis will be made easier

by the fact that many foci of disease are rarely present in caries of the spine.

Prognosis.

The prognosis of Pott's disease is always grave, *dubia ad malum vergens*. Statistics from Billroth's clinic, quoted by Oppenheim, show that out of 97 patients suffering from caries of the spine, 48, or almost one-half the number, died, 22 were cured, and 11 were dismissed as incurable. Notwithstanding this we must agree with Gowers when he says that we do not meet with any other disease of the spinal cord, even when the symptoms are most severe and of long duration, in which improvement or even recovery so frequently obtains as in the paralysis which accompanies caries of the spine. An example of this kind has been given above. There are therefore only very few cases in the beginning in which absolutely no hope of improvement may be entertained; quite as rare, however, or practically absent are the cases in which we may with certainty hope for a cure (Gowers).

In young and otherwise healthy individuals the prognosis is, of course, better than in old persons; a cure may, however, be obtained even in the latter cases. The extent of the bone disease, the degree of deformity, and the rapid or slow occurrence of paralysis are of little significance in a prognostic sense; only when paralysis makes its appearance very suddenly, in cases of dislocation of the vertebræ, the injury to the cord is usually a severe one.

If we could draw any conclusions from the symptoms as to the anatomical condition of the spinal cord, our prognosis would be more satisfactory, as we may look for recovery in cases of simple compression and œdema, even when the paralysis has been of long duration; but this, of course, would be impossible after the occurrence of true softening. Up to the present time, however, this distinction is not possible. Where the loss of function in a segment is a total one, the prognosis is not so good, for the reason that in the presence of total anæsthesia, decubitus and suppurative cystitis usually make their appearance very rapidly; but the less complete the interruption is in the affected region of the cord, the less is sensation interfered with, and the less marked are the bladder symptoms, and the more favorable consequently is the prognosis.

As regards the influence of the seat of the disease upon the prognosis, it may be said that caries of the dorsal vertebræ is the most amenable to treatment. When the cervical or lumbar enlargement is affected, the prognosis is not so good: in affection of the cervical portion of the cord, notably for the working ability of the individual

even after the caries is cured; in the lumbar region, in the first place as to life, for the reason that in this situation grave paralysis of the bladder often causes death, and then as to earning capacity, because the incomplete cure of the flaccid paralysis of the lower extremities induces more unfavorable conditions even than a spastic paralysis. In caries of the upper cervical region the probable involvement of the phrenic nerve is especially dangerous, and in disease at the extreme upper part the nearness of the medulla oblongata is a grave danger.

Early and long-continued treatment may influence the prognosis favorably. The prognosis becomes very bad, of course, in affections of the vertebræ and the spinal cord when tuberculosis appears in other parts, as the lungs or the joints, and especially upon the occurrence of miliary tuberculosis or of amyloid degeneration of the abdominal lymphatics.

Treatment.

The treatment of spinal caries and of the cord lesions accompanying it is always difficult and generally of long duration, but occasionally is a very grateful task. When called to see a case of spinal caries, our first endeavor must be, of course, to prevent an involvement of the spinal cord itself. It would of course be still better if we were able also to arrest the tuberculous disease of the vertebræ; this, however, in face of the enormous distribution of the tuberculous processes in the bones, especially in childhood, will probably remain a pious wish for a long time to come. It is, however, feasible to institute treatment at once and continue it for a long time in all cases of spinal symptoms occurring in children. This treatment would not essentially differ from that which is to be employed after a marked deformity of the spinal column and a distinct characteristic lesion of the spinal cord have already appeared. We firmly believe that by such prophylactic treatment we could prevent a large proportion of the cases of Pott's paraplegia. Unfortunately this will in most cases be impossible because of the difficulty of the diagnosis during the early stages of the disease, and the neuropathologist especially is consulted by these patients, as a rule, only after the spinal-cord lesion is already pretty far advanced.

In these cases his first duty is to enforce absolute and long-continued rest in bed. This must be continued in certain cases for many months, and we can recall cases in which this was continued even for years with very favorable results finally. When the seat of the caries is in the lumbar or dorsal spine, the patient is best placed on a perfectly flat bed, without any bolster under the head; in caries of the

cervical spine, a flat, gradually rising firmly upholstered pillow of horse hair may be employed.

We should from the beginning be careful in arranging the bed, to avoid if possible the occurrence of bedsores. For this purpose we may use, in addition to a firm but elastic horse-hair mattress, a large water pillow on which the patient's sacrum rests. The heels, which are also in danger of bedsores, should rest on small rings of some soft material. The frequent changing of the patient from one side to the other, which is usually resorted to for the prevention of bedsores, is of course not feasible in the case of caries of the spine. It is very agreeable to the patient to be changed to a fresh and clean bed at night. Next to a well-made bed the most scrupulous cleanliness will do the most in avoiding the decubitus. Drenching of the bed with urine or soiling it with fæces must be avoided; and should such an accident happen, the child's body must be thoroughly cleaned at once, and the soiled bed-clothes must be removed. It is well to lay a rubber cloth over the water-pillow, over this a clean sheet, so as to avoid the possibility of soaking the mattress with the discharges. Sponging of the sacral region with alcoholic lotions is a favorite popular method for the prevention of decubitus, and there is no objection to this if cleanliness is also practised.

In many cases simple rest in bed is sufficient to place the disease process in the spinal cord under the most favorable conditions for a cure. If the deformity is pronounced, and especially if the pain caused by the dislocation of the bones and by the compression of the roots of the spinal cord is very severe, we may, besides the simple flat position, also try extension of the spinal column. This may be accomplished in the best manner, in the case of cervical caries, by the application under the chin of a sling, the bed being lowered at the foot in order to obtain counter-extension by the weight of the body; or we may attach a weight to the lower extremities. This extension acts particularly well in cases in which severe pain from compression of the nerve roots is present, as in tuberculosis of the upper cervical vertebræ. Corsets of felt or plaster-of-Paris are best avoided, as being unnecessary.

It is of course important to keep the general health up to the highest point. For this purpose we must supply good, fresh air. When the circumstances of the patient permit, we should make use of two sick-rooms, which may be alternately used, perhaps one during the day, the other during the night. The patient may also be taken into the fresh air in a well-made ambulatory. The food should be simple but nourishing. Cod-liver oil is especially to be recommended. Of medicines proper, preparations of the iodide of iron are

of the greatest benefit. Derivatives, which formerly were so greatly praised, particularly the more energetic ones, such as the actual cautery and issues, are best forgotten. In caries of the cervical vertebræ, however, we may still make occasional use of the horse-hair issue. But all such measures are weakening and are liable to depress the patient and increase the danger of decubitus. Other tuberculous affections, in case they should appear, are to be treated *secundum artem.*

By perseverance in the method above described, which may and should be varied in its details according to each case, we shall often experience the satisfaction coming from a perfect cure of the bone and cord affection. In many cases the grave complications of decubitus and of cystitis will not appear at all, the affection of the cord confining itself to a spastic paraparesis. Should these complications occur, however, they are to be treated according to the general rules of surgery. A cure may quite frequently be obtained even in cases of nearly complete paralysis, extensive decubitus, and great deformity of the spinal column, by proper careful nursing and attention to the nutrition of the patient. This is, however, not always the outcome, and unfortunately in one-half of the cases death takes place from decubitus and cystitis, or with especial frequency from other tuberculous affections, or rarely from the disease of the cord itself. For the further treatment of these cases special directions cannot be given. Frequently we must be content to moderate the patient's pain by the heroic employment of narcotic remedies. The demands of nursing in these cases are, as may readily be seen from the above, quite imperative, and it is rare that these can be met in private practice, so that usually a removal of the patient to a well-managed hospital is indicated. For a time direct surgical intervention in the treatment of Pott's paraplegia was regarded with great favor, but the experience of late years has taught us that our hopes in this direction were greatly exaggerated, and that an operation is rarely of any benefit, if indeed a fatal result is not hastened by it. It is very difficult to decide when to operate in these cases; indeed it is only in cases accompanied by marked and rapidly increasing respiratory disturbances that the indication is wholly in favor of an operation, which is the sole means of rescue here. It is certain that we cannot expect a cure through operative interference in a great number of cases, leaving possible complications altogether aside. The most frequent seat of the lesion in the bodies of the vertebræ renders the access to the diseased region very difficult. The case is of course hopeless, and an operation would only make the condition worse, if, as frequently happens, a number of vertebræ are destroyed. In cases of acute com-

pression of the cord an operation is contraindicated unless we have to deal with the rare condition of the presence of a loose sequestrum. The operative treatment of those truly rare cases in which the tuberculous affection has passed through the pia mater to the cord does not offer any chance whatever, and Chipault advised that the dura should never be opened in caries of the spine, for the reason that any subdural processes which may be present cannot in any event be removed, and the danger of the operation is materially increased.

We know, however, much better and with greater certainty when an operation is not indicated than when an operation may be attempted. The first cases are in the great majority, those of the second category are few. We follow here the statements of Chipault, which are based on a ripe personal experience and on a thorough study of the literature.

The operation is contraindicated: 1. When a large number of the vertebral bodies are involved, and especially when the arches are also attacked. A full removal of all diseased portions would in this case be impossible without so injuring the spinal column that it would never be possible for the patient either to walk or to stand. 2. When other organs are also affected by tuberculosis, especially in pulmonary phthisis. We must consider here that a monolocular invasion of the body by tuberculosis is a rare occurrence. 3. When the cord itself is directly affected by the tuberculosis.

The operation is indicated: 1. In cases in which the disease of the bones is confined to the arches, and in which this disease condition is emphasized by the appearance possibly of an abscess on or at the side of the spinous processes. When such a circumscribed posterior disease has led to disturbance of the functions of the cord, its removal by operation will naturally exert a very favorable influence. 2. When the compression of the cord is caused by a cold abscess which is connected with another lying outside of the spinal column. The operation in these extraordinarily favorable cases consists only in incising the external abscess, and perhaps also in removing the diseased portion of bone. 3. There are cases met with in dorsal compression of the cord in which the paralysis is a flaccid one from the outset (Babinski). It seems as if the fungosities in these cases only produced a simple compression of the cord and that they are in themselves usually of little moment. We might therefore advise an operation in cases of tuberculosis of the dorsal region of the spinal column when a flaccid paralysis has been present from the beginning. Chipault has operated successfully in such a case. 4. There are cases of tuberculosis of the spine in which the bone affection has healed and the fungoid masses have become cicatrized, but

will be found under that head; but the rare solitary tubercles of the cord will be discussed here.

Tumors of the Spinal Column.

Pathological Anatomy.

The so-called vertebral tumors may spring from the bones of the spine or from the surrounding tissues. In the latter case they implicate the spinal column only secondarily. Of these forms of tumor two are of special importance. First, sarcomata which may start, for instance, from the muscles of the spinal column or from the prevertebral lymphatic glands and thence very gradually invade the vertebral canal either after destroying the bones or through the intervertebral foramina. Their sites of predilection are the lower portions of the spinal column, particularly the sacrum, where they not rarely cause, in addition to the pains due to the erosion of the bones, compression of the pelvic viscera and, through lesion of the sacral plexus, pains in the sciatic nerve and atrophic paralyses in the distribution of the sacral nerves. Later on, after perforation of the bones, they may also involve the cauda equina directly. On rectal examination these tumors can often be recognized early.

The second form is aneurysm of the aorta, which perhaps cannot be properly included among the tumors but may produce symptoms very similar to those due to neoplasms of the vertebral column. While very uncommon in the case of abdominal aneurysm, it is more frequent to find a thoracic aneurysm extending especially posteriorly and medially, eroding a vertebra, and finally compressing the cord directly, after having caused violent pains by irritation of the posterior roots at their points of emergence through the bone. The symptoms of aneurysm of the thoracic aorta would be at first violent pains in the back, especially with movement, such as in coughing or sneezing, but also after prolonged recumbency at night; these pains are moreover markedly increased by movements which raise the blood pressure. Then there is a very pronounced rigidity of the back. Subsequently there will be in addition severe lancinating intercostal girdle pains due to the pressure of the aneurysm upon the posterior roots. Finally spastic paralysis of the legs and vesical and sensory disturbances follow. Death is almost invariably due to rupture of the aneurysm, and the hemorrhage may then take place into the spinal canal. The diagnosis is possible after that of the aneurysm has been made. Moreover, aneurysms of the descending aorta, even without lesion of the spinal column, may cause the most

violent girdle pains by direct pressure upon the intercostal nerves; not rarely these intercostal neuralgiæ are for a long time the only symptom of the aneurysm.

Of the true tumors of the vertebral bones the following come under observation: carcinoma, sarcoma or osteosarcoma, myxoma, exostosis, especially multiple exostosis, and finally echinococcus.

Vertebral carcinoma is said to occur primarily, though in very rare cases; in the greatest majority of cases, however, it is of metastatic origin. Probably it is most frequent after cancer of the breast, more rarely after cancer of the stomach or of the uterus. Owing to this primary seat of the tumor, vertebral carcinoma is decidedly more frequent in women than in men. Its most frequent location is the middle portion of the dorsal spine, more rarely the upper lumbar or the lower cervical vertebræ are affected. The tumor, moreover, even when the symptoms point to a circumscribed location, often involves a large number of the vertebræ; in one of the writer's cases marked by pains in the distribution of the lower dorsal roots, all of the vertebræ from the first cervical to the sacrum were infiltrated with cancer and only a very thin shell of bone was left. In most cases the primary seat of the carcinoma is in the vertebral bodies; more rarely it springs from the lateral portions, narrows the intervertebral foramina, and compresses the nerve roots. In quite exceptional cases it involves also the spinous processes, so that the tumors may be recognized by their thickening and deformity. When one or more vertebral bodies are completely destroyed, they may fall together; occasionally this may result in a gibbus, more frequently in a crowding together of the vertebræ, leading to a shortening of the spine and hence of the individual. With such extensive destruction as above described we can only marvel that an erect posture is at all possible. As a rule, in vertebral carcinoma the cord proper is long spared, even when shortening of the spine occurs it often remains uninjured; a sudden gibbus formation, however, is more likely to cause immediate compression, but frequently lesions of the nerve roots alone exist. Not rarely the extradural adipose tissue ultimately undergoes carcinomatous infiltration; in that event the cancer may surround the cord for long distances in the form of a complete or half cylinder, the dura being but rarely penetrated.

Sarcoma and osteosarcoma of the vertebral column probably spring in most cases from the periosteum. They differ from carcinoma in being even more malignant; they grow more rapidly and oftener cause considerable deformities and gibbosities, which usually disintegrate rapidly.

Exostoses—according to Marchand these are generally the so-

important than the signs of irritation. This is due to the extensive anastomosis of the various roots, in consequence of which every muscle and every cutaneous region is supplied by a number of motor and sensory roots, so that many of these must be destroyed to produce paralysis, while the lesion of a single root suffices to cause symptoms of irritation. Among the rarest are circumscribed, distinct anæsthesiæ corresponding to the distribution of the roots, before the cord itself is involved. More frequently we find paretic symptoms and trophic disturbances in the muscles, associated with the reaction of degeneration; there is a very active fibrillary twitching. In one case under my observation the atrophy and paralysis were absolutely parallel, as in typical spinal progressive muscular atrophy. In these cases we observe not rarely also trophic disturbances of the skin, such as parchment-like thinning and glossy skin, so called. These phenomena, of course, can be demonstrated only on the arms and legs, with corresponding location of the carcinoma.

The *cord symptoms* are those of compression at a certain level. Not until this occurs are the motor and sensory paralyses as a rule clearly marked; if it is approximately complete we find extensive paralysis and anæsthesia in all the regions innervated by the cord below the point of compression. The type of the symptoms, therefore, is paraplegia which develops gradually, or rarely acutely by the sudden displacement of the vertebræ and gibbus formation or by myelitis; quite exceptionally only one-half of the cord may be implicated in the beginning. Of course we cannot here enter in detail upon the extent and distribution of the paralyses and anæsthesiæ which vary with the level of the tumor, nor upon the changeable condition of the bladder and rectum, of the reflexes and trophic disturbances, especially bedsores; the most essential points of these will be mentioned under tumors of the meninges. One more symptom may be mentioned in the present connection, one which was first described with vertebral carcinoma, viz., the occurrence of severe pains in the paralyzed limbs, usually the legs, Cruveilhier's paraplegia dolorosa. When the legs in these cases are at the same time anæsthetic, we may also speak of an anæsthesia dolorosa.

Diagnosis.

The diagnosis of a vertebral tumor—and here we refer first only to carcinoma and sarcoma—may be very easy under certain circumstances and when all the symptoms are clearly marked, especially when a primary tumor has been demonstrated in some other part of the body. As a rule, however, it is difficult, and can be made posi-

tively only in the later stages of the disease. When there are at first, as in typical cases, only vague pains in the back and along the spinal column, it will evidently be impossible to express a decided opinion as to the case. When symptoms of a root or cord lesion are gradually added, the case may still be one of simple myelitis or of syphilitic meningomyelitis. Only when distinct signs appear in addition in the bones, particularly the development of a gibbosity or other deformities of the vertebral column, we are nearer a solution; at any rate we are then forced to assume that there is a primary disease of the spine with gradual involvement of the roots and cord. But the most frequent disease of the kind is caries of the spine and from this the tumor is to be first of all differentiated. This, however, is more easily said than done. Most of the differential factors are vague and in a concrete case of doubtful nature. While tuberculosis is usually a disease of early life, it is not very rare in middle and even advanced age, especially in the spinal column. Carcinoma, it is true, occurs usually only late in life, but sarcoma may develop even in early childhood. In a doubtful case, tuberculosis of other organs would be in favor of caries. According to Gowers, the root symptoms in carcinoma of the spine are much more violent and prolonged than in caries, but this is not always true. A rapid development of paraplegia is said to be far more frequent in carcinoma and to be due to acute myelitic softening; but the same symptom is not particularly rare in caries, especially of the cervical spine, in consequence of sudden displacement of the vertebral bodies. Gibbus formation is much more frequent in caries than in carcinoma, but shortening of the spinal column is more common in the latter. Unilateral lesion occurs both in caries and in carcinoma in rare cases. The demonstration of a distinct spinal tumor would be decisive; this is most likely to succeed in the case of sarcoma, especially by rectal examination when the neoplasm is located on the sacrum. The diagnosis is also nearly certain when the symptoms of spinal disease and compression of the cord develop in connection with an existing carcinoma, *e.g.*, of the breast, or after its operative removal. It is, however, to be noted that in cases of carcinoma also simple toxic forms of myelitis and neuritis may develop, and that Schlesinger has reported the case of a patient with carcinoma in whom an associated cord and vertebral lesion was due to tuberculosis. In a parallel case of the writer's the presence of a retropharyngeal abscess decided the diagnosis in favor of tuberculosis.

Not rarely the sole symptom of a vertebral tumor, for a long period of time, is a severe intercostal neuralgia. Simple intercostal neuralgia occurs almost exclusively in hysterical and anæmic women,

Here again those developing intradurally surpass in numbers those of extradural origin. Of fifty-eight meningeal tumors collated by Horsley twenty were extradural, thirty-eight intradural. Extradural and above all intradural tumors are more rarely of metastatic than of primary origin; more frequent is a simultaneous development of a tumor of the cord and of some other part of the body; usually they are solitary and in rare instances only are they multiple, but in the latter case they may be innumerable, particularly sarcomata of the pia. Of primary extradural tumors we may meet with lipomata developing in the extradural adipose tissue, sarcomata springing from the periosteum of the spinal column or from the outer surface of the dura; in isolated cases enchondromata have been observed, and echinococcus cysts are somewhat more frequent. Secondarily we find carcinoma in the extradural adipose tissue without involvement of the spinal column, also sarcomata and teratomata. These metastatic extradural tumors form not rarely very flat deposits upon the dura, which they surround for considerable distances in the form of a completely closed cylinder or one open towards the vertebral bodies.

Within the dura are to be observed, springing from the inner surface of the dura, from the ligamentum denticulatum, from the arachnoid, or from the outer surface of the pia, fibromata, fibrosarcomata, sarcomata, myxomata, particularly also vascular tumors such as angiosarcomata and true angiomata. In the region of the lumbar enlargement and in connection with spina bifida we may also find lipomata. Multiple neuromata have been observed on the roots of the cauda equina. In the arachnoidal space we may meet with cysticerci which are usually multiple, and more commonly solitary echinococcus cysts. True solitary tubercles of the meninges are not rare.

Tumors of the meninges, even the different forms of sarcoma, do not in general destroy the meninges themselves. On the whole it is rare to find an extradural tumor perforating the dura or an intradural tumor piercing the pia and extending into the cord. This of course is not the invariable rule, and the writer was once able, in a case of multiple sarcomatosis of the pia, to demonstrate clearly the perforation of the tumors into the cord. Tumors of the meninges, especially the intradural varieties, exhibit in general considerable resemblance in form. This is due to the fact that they rarely perforate the bone and hence must adapt themselves to the space in which they lie, *i.e.*, the spinal canal, and here first to the cavity between the vertebræ and the cord. They rarely attain a large size; at first they are approximately spherical, later they increase in length rather than in breadth so that they ultimately become cylindrical. They are usually situated laterally or postero-laterally, very rarely in front of the cord,

by the more or less marked compression of which they make room for themselves. If they perforate the bone—an uncommon occurrence except in the case of echinococcus cysts—they may of course become very irregular in form and quite large. In a case operated upon by the writer, in which the tumor could not be found at the time, it had reached the size of an apple in the trephine opening when death occurred. At the cauda equina alone do the tumors find room for greater development even when the spinal canal remains closed. The shape of metastatic extradural sarcomata and carcinomata has been discussed above.

A meningeal tumor may produce its effects in the direction of the bone, the roots, or the cord. As a rule the bone lesions are the last to appear and only rarely do they attain any importance. Usually the lesions of the roots appear first, then those of the cord. An extradural flat tumor may involve the roots for a long time and to a considerable extent without implicating the cord, while an intradural tumor will attack the cord more rapidly. When of great width an extradural tumor will of course also compress the cord early. The roots may be both compressed and infiltrated; often they oppose considerable resistance to the tumors so that even in extensive injury well-preserved root fibres are met with.

The cord may be simply compressed without material impairment of its structure, so that after the cessation of the pressure its recuperation may be almost perfect; it may also be infiltrated with œdema or be softened throughout. The last-mentioned condition is most likely to occur when the tumor grows rapidly and is rather hard at the same time, still it is probable that some part is played by true inflammatory processes due to tumor toxins or by ischæmic softening.

In the substance of the cord proper we meet with glioma, sarcoma, angiosarcoma, solitary tubercle, and cysticercus. The different forms of sarcoma almost never originate in the substance of the cord, but proliferate into it from the pia, either from its inner surface or from without after its perforation. Glioma arises in the cord itself and frequently in its vicinity, particularly the posterior periphery of the central canal; tubercles spring from the vessels of the cord and according to Gowers often have their primary seat in the gray substance of one side or else in the posterior horns (Schlesinger). Cysticercus, which is rare, may of course have its seat anywhere in the substance of the cord. As a rule, intramedullary tumors too are primary and solitary, only tubercles occasionally occur in numbers. Tumors of the cord, like those of the brain, may either be sharply demarcated from the normal structure or they merge gradually into it—they infiltrate the tissue, as it is called. The sharply demarcated

neoplasms are sarcoma, tubercle, and cysticercus; the cord surrounding them is usually softened for a certain distance. Infiltrating growths are gliomata; they may extend over long distances, especially in the longitudinal axis of the cord, and far into the caudex cerebri; in that case they break down in the centre and present the well-known picture of syringomyelia, which corresponds in no way to that of the other tumors of the cord and therefore is not discussed in this section. Sometimes, it is true, gliomata of the cord occupy a more circumscribed site, are firmer in consistence, and compress the cord like any other tumor.

Intramedullary tumors are usually small, and spherical or cylindrical in shape; they extend longitudinally rather than transversely; only gliomata sometimes enlarge the diameters of the cord so that, as Gowers noted, the margin of the occipital foramen may cut directly into the cord.

Since many if not most of the cord tumors spring from the pia we observe again first root symptoms, then medullary symptoms; in tumors having primarily a central location the order may be reversed and in consequence some peculiarities of the morbid picture may result, which will be discussed at greater length hereafter.

Symptoms.

The essential feature in the morbid picture of both extra- and intramedullary tumors of the cord is a slow compression of the latter and its roots. Hence tumors of the meninges and of the cord do not differ in their symptoms very markedly from those of the spinal column when the latter have developed sufficiently to produce medullary symptoms along with the root symptoms. The first to appear are probably always symptoms of disease, especially irritation, of the roots. In intravertebral as in vertebral tumors the dominant symptoms are those of irritation of the sensory roots, *i.e.*, the pains. Contrary to what we find in vertebral tumors, the pains in meningeal tumors are at first almost invariably unilateral, and this because these tumors usually develop on one side of the cord; only later, after a marked lesion of the intervening cord has resulted, do they involve also the opposite side. Of course, a tumor situated exactly in the median line posteriorly on the cord may cause bilateral pains from the beginning..

The seat of the pains depends obviously upon the level at which the tumor is located; the most frequent are intercostal, then brachial, lumbar, and sacral pains; these will be discussed at greater length later. The only characteristic features are that the pains never

correspond to the course of a peripheral nerve, but are always in harmony with the distribution of the spinal roots, and that the pressure points of a true peripheral neuralgia are absent.

Otherwise the pains present the typical neuralgic character—they are tearing, lancinating, ever recurring at the same point; a local sensation of intense painful burning, which renders even the weight of the clothing irksome, is especially frequent. In the latter case the skin is also hyperæsthetic. Herpes zoster seems to be less common in tumors of the meninges than in those of the vertebræ. The pains are usually extremely violent and prolonged, still painless periods occur even with steadily growing tumors; this is probably the case when the neoplasm has completely destroyed one posterior root and requires some time to reach the next. This is more apt to occur in the dorsal than in the cervical and lumbar regions, because in the former the several roots are farther apart, while in the latter there are hardly any intervals.

In the case of intravertebral tumors, too, the irritative symptoms presented by the motor roots are less pronounced than those of the sensory roots; otherwise they do not differ from those described under the head of vertebral tumors. We meet therefore with more or less painful cramps and persistent spasms of isolated muscles, *e.g.*, those of the abdomen, or those of the neck and nucha.

Paralytic symptoms dependent upon a lesion of the roots alone without that of the cord occur much later, for the reasons discussed above. Besides, contrary to what takes place in the case of irritation, the motor root symptoms predominate, *i.e.*, pareses, atrophies, reactions of degeneration, and fibrillary twitchings; the electrical disturbances are probably the last to appear. For the reasons given above, several roots must be affected in these cases, and especially marked anæsthesiæ—like plexus paralyses in their arrangement—can result obviously only when at least three roots are largely involved. This is possible, without coincident implication of the cord, solely in cases of the above-described flat but very long extramedullary tumors. Moreover, the anæsthesiæ and motor paralyses in all these cases will be confined to the distribution of the roots which are directly affected by the tumor. All these facts were clearly illustrated by one of the writer's cases of meningeal tumor which had obviously sprung from the lower lumbar roots and in which, at a time when several roots were unquestionably already involved, not the least anæsthesia could be found, and long after the onset of paralytic symptoms in the distribution of the tibialis posticus and peroneal nerves the muscles neither became atrophied nor showed electrical disturbances, so that hysteria was repeatedly suspected.

The case presents quite a different aspect when the tumor involves the cord. The anæsthesiæ and paralyses immediately become more extensive; for, as conduction is interrupted, they implicate regions innervated by the cord below the actual seat of the lesion. Since the tumor, as we have seen, springs almost always from one side between the cord and bone, it will, as a rule, first injure one-half of the cord and thus the symptoms of a unilateral lesion, those of Brown-Séquard's paralysis so called, will be manifested. In typical cases of the kind we then find on the side of the tumor, in the region situated below it, paralysis with usually increased reflexes and loss of the muscle sense; at the level of the tumor, an anæsthetic and a hyperæsthetic zone; on the opposite side, anæsthesia in the entire cord region situated below the tumor. In case of a growing neoplasm, however, the tumor will soon involve the opposite side, when total paraplegia and anæsthesia will occur in the region below the compressed point. In gradual compression of the cord first the muscles of the feet, then the knee, then the hip are paralyzed and the anæsthesia spreads in like manner in an upward direction; but not rarely the paraplegia is complete to the level of the tumor, while the anæsthesia is clearly marked only on the legs. Of course when the whole of the transverse section is interrupted special symptoms will still be present at the point where the tumor has its seat; in this locality the root irritation advances steadily and will produce one new symptom after another, especially in an upward direction. At this level there will be additional neuralgias and hyperæsthesiæ and, particularly when the cervical or the lumbar enlargement is implicated, marked atrophic paralyses associated with electrical disturbances of the muscles. The paralysis below the lesion, however, will at first not be combined with distinct trophic disturbances of the muscles. On the other hand, according to the law of eccentric projection of the sensations, painful sensations may occur even in the totally paralyzed and possibly also completely anæsthetic extremities (anæsthesia dolorosa). Ultimately the meningeal tumor will also involve the bone—at first perhaps only the internal periosteum of the spinal column—with resulting painful rigidity, pain on motion, coughing, or sneezing, and the corresponding regions will besides be sensitive on pressure and percussion. Even mere sitting up may cause pain at the site of the tumor. At the same time there will be vesical and rectal paralyses, bedsores develop, and the patient finally succumbs to general marasmus unless the tumor has caused death previously by reason of its seat.

Having now described the typica course of an intravertebral tumor, we must dwell upon some deviations from the rule. First of

all, the paraplegia may not be the gradual result of a unilateral lesion, but may appear acutely in consequence of œdema, ischæmia, or sometimes perhaps of a true myelitis. Other differences may be due to the development of the tumor extradurally, intradurally, or in the cord proper. These variations may not be very marked, but the above brief description applies most closely to meningeal (intradural) growths which are the most frequent. The description is true also of medullary tumors which begin upon the pia, while truly central neoplasms may run their course at first entirely without pains and consequently simulate the course of a simple myelitis, or that of a simple progressive muscular atrophy when they develop, *e.g.*, in one, or more frequently in both anterior horns and follow the direction of the longitudinal axis, or that of syringo-myelia when they develop simultaneously in the anterior and posterior horns (Schlesinger).

Finally, extradural tumors will obviously at an earlier period give rise to vertebral symptoms, to circumscribed periosteal sensitiveness, and to rigidity of the back; these in fact may be the first symptoms as in vertebral tumors.

We must next consider those variations in the morbid picture which depend upon the accurate localization of the symptoms of irritation and interrupted conduction and are due to the difference in the level of the tumor; these are very important and of decided practical interest, since they alone can furnish us information as to the seat of the neoplasm. Roughly we may differentiate tumors of the cervical, dorsal, and lumbar cord, and of the cauda equina. In all cases there are root and eventually bone symptoms corresponding to the level of the tumor; below this, such disturbances of motility, sensibility, and of the reflexes, etc., as are dependent upon the interrupted conduction of the cord from the segments situated caudad of the lesion.

Tumors of the Upper Cervical Cord.—At first perhaps hemiplegia of the arm and leg on the side of the tumor, differing from central hemiplegia in the non-implication of the facial and hypoglossal nerves, eventually with anæsthesia of the opposite side (Brown-Séquard). Later, spastic motor paresis or paralysis of all four extremities. Preceding the hemiplegia and paraplegia, pains and perhaps circumscribed anæsthesiæ in the cervical plexus, also in the distribution of both supraclavicular, the occipitalis minor, the auricularis magnus, and possibly also the occipitalis major nerves; atrophic paralyses in the region of both sternocleidomastoids, the trapezius and scaleni, the deep anterior cervical, and the superficial and deep nuchal muscles. The paraplegic stage is apt to be of very short duration, as the affection of the phrenic nerves soon terminates fatally.

Tumors of the Cervical Enlargement.—First, atrophy and flaccid paralysis, perhaps pains, in the arm corresponding to the side of the tumor, spastic paralysis of the leg of the same side, anæsthesia of the opposite extremities and of the opposite half of the trunk. Later, spastic paraplegia and anæsthesia of both lower extremities and anæsthesia of the trunk to the level of the second rib, atrophic flaccid paralysis of the upper extremities. When the tumor involves the entire cervical enlargement, all the muscles of the upper extremities are affected with total anæsthesia of both arms; when only the upper half is directly implicated, there is atrophy of the upper arm and shoulder muscles with spastic paralysis of the hand and fingers, and anæsthesia as before; when the lower half of the cervical enlargement is alone affected, the shoulder and upper arm are intact, there is flaccid atrophic paralysis of the forearm and hand muscles, and the anæsthesia is confined to the ulnar half of the upper extremities. When the first dorsal root is directly involved, there are myosis and narrowing of the palpebral fissure, both first on one side, then on both, and eventually, pains in the shoulders and arms.

Tumors of the Dorsal Cord.—Brown-Séquard's paralysis is usually pronounced. Later, there are spastic paraplegia of the lower extremities and of the abdominal muscles, and anæsthesia of the trunk to a level corresponding to the seat of the tumor. At first unilateral, then complete girdle pains and hyperæsthesiæ around the thorax or abdomen, not corresponding to separate intercostal spaces but with more horizontal limits. Atrophic paralyses cannot be demonstrated in the intercostal muscles corresponding to the level of the tumor, but may eventually appear in the abdominal muscles.

The tendon and skin reflexes, the condition of the bladder and rectum, and the trophic disturbances of the integument are in general the same in the different locations of the tumor thus far described. In the Brown-Séquard stage the tendon and skin reflexes are at first increased on the side of the tumor, and they become so on both sides when paraplegic symptoms set in. In these cases the increase is very marked; patellar and Achilles clonus and often pronounced so-called spinal epilepsy are present. But gradually, when the lesion of the transverse section becomes more and more nearly total, the tendon reflexes in particular diminish, and with complete transverse interruption they are altogether absent, whatever the seat of the lesion. At the same time, unless adhesions have previously formed in the joints—an occurrence especially common in flexion contractures—the spastic paresis again becomes flaccid, and while the muscles, particularly those of the lower extremities, do not present a degenerative atrophy, yet they shrink and their electric irritability may be much

reduced quantitatively. The skin reflexes, which always exhibit great individual variations, do not present, even under these circumstances, so constant a relation as the tendon reflexes; the plantar reflex in particular sometimes persists when a total transverse lesion must be assumed to be present for other reasons.

The state of the bladder varies greatly in different cases. At first there is dysuria, the patient being obliged to stand or sit long and to strain; by degrees micturition becomes impossible. At first there is also not rarely a greatly increased and oft-repeated desire to pass water (pollakiuria)—and this assumes, as it were, an imperative character—the patient must yield to it at once if he would avoid soiling himself. Later, the so-called intermittent incontinence may develop—the evacuation of the bladder is no longer subject to the control of the will, but when a certain degree of fulness of the viscus has been reached the sphincter relaxes and the detrusor contracts reflexly, the vesical contents are voided in a powerful stream. When there is no anæsthesia the patient of course is conscious of such evacuation. After the reflex activity of the detrusor has also become impaired the sphincter is forced open only when the bladder is completely filled and the urine dribbles away from the full bladder—ischuria paradoxa. When the transverse interruption is complete the detrusor and sphincter, as well as the entire reflex activity of the bladder, are wholly paralyzed, even when the lesion occupies a high level. Even then the urine never dribbles off directly as it reaches the bladder from the kidneys; the elasticity of the sphincter can always still retain a certain quantity of urine, so that the escape does not ensue until the pressure exceeds the elastic occlusion, that is, after some degree of filling of the bladder. Then the viscus may also be emptied by mechanical pressure, and even slight changes in the latter, such as coughing, intestinal movements, and sitting up expel some quantities of urine from the bladder. The elastic closure of the bladder is much weaker in women than in men. In nearly all cases of serious vesical paralysis the occurrence of cystitis, pyelitis, and nephritis is not long delayed, and these complications are very frequent causes of death. From the onset there is usually constipation, which becomes more obstinate as the transverse lesion extends. In a certain stage, voluntary evacuation may already have become impossible even with the aid of abdominal pressure, though the intestine continues to act reflexly and the sphincter still grips the finger introduced into the anus. With total transverse lesion the sphincter ani is likewise completely paralyzed and no longer closes around the finger. Nevertheless the fecal masses which are usually very dry and hard are retained, but every few days a portion of the mass is mechanically forced out by the after-crowding fæces;

the patient does not soil himself, however, unless he suffers from diarrhœa.

Bedsores occur with cord tumors located at a high level when the anæsthesia is marked, that is, when the transverse lesion is approximately total. In other respects they present the same features as in tumors of the lumbar cord.

Tumors of the Sacral Cord.—Atrophic paralysis of the muscles of the leg and foot, of the posterior surface of the thigh, of the gluteal and peroneal muscles, first on one side, then on both sides; anæsthesia affects the foot, the posterior surface of the leg and thigh, the perineum, and the genitals. Total paralysis of the bladder and rectum. Impotence. Loss of the Achilles tendon reflex, with possible preservation of the patellar tendon reflex. Initial pains in the distribution of the sacral plexus of the side first affected. Bedsores and cystitis develop early.

Tumors of the Lumbar Portion of the Lumbar Enlargement.—Initial unilateral pains in the distribution of the lumbar plexus, hence on the anterior and inner side of the thigh and leg and in the fold of the groin. Atrophic paralyses likewise first in the region of the lumbar plexus, hence in the ileopsoas, quadriceps, adductors, and tibialis anticus. Very rarely also anæsthesiæ in the same region with isolated root lesions; with the beginning of the cord lesion, however, total anæsthesia occurs in the region of the lumbar plexus; in that of the sacral plexus, partial anæsthesia, perhaps only to temperature and pain. This grouping is an almost positive proof of development of the tumor at the lumbar cord. Brown-Séquard's symptoms may also be produced by a unilateral affection of the upper part of the lumbar enlargement. Later there are complete paralysis and anæsthesia of the lower extremities, but only in the distribution of the lumbar plexus, associated with atrophy and the reaction of degeneration, on the legs and feet eventually with spastic symptoms. Achilles clonus may be combined with them, while the patellar reflexes are absent. Vesical and rectal paralysis is not so marked in the beginning as in the case of sacral tumors.

The lumbar enlargement in particular is very short and its roots are closely crowded together. Tumors of the meninges, therefore, will only for a short time produce pure sacral or lumbar symptoms. The differences are usually from the first indistinct or become early obliterated; we then find simultaneously symptoms pointing to the entire lumbar enlargement, while the pains occur in the distribution of the highest implicated roots.

Tumors of the Cauda Equina.—It is obvious that there can be no essential clinical differences between tumors of the cauda equina and

those of the lumbar enlargement, since the roots of the former are nothing but processes of the lumbar enlargement, and the roots and their segments have no separate functions. Nevertheless we can discover some differential points. While the manifestations of all other meningeal tumors begin with root symptoms of one side—the transition to the other side is somewhat retarded by the cord itself—in tumors of the cauda bilateral symptoms are generally present from the beginning. Brown-Séquard's symptoms of course cannot be produced by a caudal neoplasm. Should a caudal tumor begin exceptionally with unilateral symptoms, which evidently is not impossible, it will take longer to extend to the other side, *i.e.*, to produce paraplegic symptoms. Finally, the paraplegia in caudal tumors is not as a rule so symmetrically distributed over both lower extremities as when it is caused by a cord lesion; a tumor of the cauda attains about the same symmetry as does multiple neuritis.

Typical reaction of degeneration in extensive muscular regions will in doubtful cases indicate rather a caudal lesion; affections of the lumbar enlargement are perhaps more likely to cause partial reaction of degeneration, but on the other hand produce more marked fibrillary contractions. The pains are especially violent and widespread in caudal tumors, probably because so many sensory roots are in close juxtaposition at this point, and they are particularly severe in the sacrum and ischium. The disturbance of the vesical, rectal, and genital functions will not differ from that in tumors of the lumbar enlargement. An important differential factor consists in the following circumstance which has been learned from practical experience. Tumors of the cauda equina, like traumatisms in this region, produce at first only symptoms in the sacral plexus and do not involve even the lowest lumbar roots. This is not easily understood in the case of trauma which, we are forced to assume, would be more likely to injure the fibres of the lumbar plexus situated on the outer side close to the bone, than those of the sacral plexus occupying the centre. In the case of tumors we must suppose that they develop by preference in the median line. Be this as it may, at any rate a caudal tumor may manifest itself for a long time by purely sacral symptoms, while a new growth of the lumbar enlargement, as we have seen, soon affects, whenever it involves the cord at all, both the lumbar and the sacral plexus. Of course this is not an invariable rule applicable to every case; for a tumor in the sacral portion of the lumbar enlargement may at first produce functional disturbances only in the sacral plexus, and this the more readily because, according to a law of general application, it usually spares the roots of the lumbar plexus which pass by it; and moreover, a tumor of the cauda may occasionally injure not

only the sacral but all the lumbar roots, excepting perhaps the first, before they emerge from the cord, and without touching the conus. At all events it is probably safe to assert positively that when the symptoms begin with disturbances in the distribution of the lumbar plexus, followed subsequently by disturbances in the sacral plexus, the tumor is one of the lumbar enlargement and not one of the cauda. This diagnosis is strengthened when the sacral symptoms present at first such peculiarities as are caused by interrupted conduction below the actual seat of the tumor (spastic symptoms in the leg and foot), or when the lumbar symptoms merely predominate over the sacral, about in the way observed by Bruns and by Allen Starr, the anæsthesia being complete in the region of the lumbar plexus and partial in that of the sacral plexus.

The diagnosis is also absolutely certain when there is marked sensitiveness on percussion below the second lumbar vertebra, since the conus terminalis ordinarily reaches only as far as the middle of this vertebra. In short if several of the above-enumerated differential factors concur, especially the positive signs, such as sensitiveness on percussion of the lower lumbar spinous processes and of the sacrum, or spastic symptoms in the leg muscles, the differential diagnosis between caudal and lumbar-cord tumor may be positively made. In less favorable cases this is not possible, and hence if an operation is undertaken it is better to make the opening large enough to permit inspection of the lumbar and caudal regions that may possibly be involved, and as the lumbar enlargement is quite short this will not necessitate a very great opening.

Course.

The course, particularly of tumors of the meninges, is chronic; occasionally the affection may be prolonged over several decades, but usually it lasts two to three years. The root symptoms, especially the pains, are often alone present for several years, the motor root symptoms not developing until later. As soon as injury of the cord proper is superadded the progress is usually more rapid; in the cervical cord death may suddenly result from asphyxia, otherwise it is usually the consequence of complications in the bladder, of bedsores, or of general marasmus. The duration depends pre-eminently upon the rapidity of growth of the tumor; malignant neoplasms usually increase more quickly, and it is probably in consequence of the more frequent occurrence of metastatic carcinoma and sarcoma in the extradural adipose tissue that extradural tumors have a more rapid course than intradural tumors, the proportion being one year and one month as against two years and three months.

Diagnosis.

The diagnosis of an obscure cord tumor will always remain one of the most difficult problems. Two things are necessary to make this diagnosis possible. In the first place we must have some information—the more accurate the better—of the course of the disease, for in typical cases the characteristic features are the initial unilateral root symptoms with unilateral and later bilateral cord symptoms. When this sequence cannot be learned and we are face to face with the complete symptom complex, the diagnosis will often not be a positive one. In the second place the symptoms must have reached a certain development before the diagnosis is possible. As long as the morbid picture is composed solely of neuralgic symptoms the positive diagnosis of a tumor is out of the question. Under such circumstances it may even be difficult to differentiate a simple intercostal neuralgia or the pains produced by disease of the heart or the great vessels, though at times the diagnosis of a root lesion from a peripheral neuralgia may be possible from the localization of the pains corresponding to root regions and the absence of sensitive pressure points. Hysterical neuralgiæ and those caused by a tumor might be differentiated by the fact that the latter are incessant and rebellious, so that even in this stage we would be led to suspect a serious organic disease. This suspicion will of course be verified when paralyses, and particularly circumscribed muscular atrophies with electrical disturbances, are superadded to the neuralgiæ. But in tumors of the dorsal cord such trophic disturbances cannot be demonstrated, and, moreover, at this early period it is impossible to make a differential diagnosis between simple forms of neuritis of the plexus in question and prevertebral tumors, *e.g.*, neoplasms in the pelvis and at the anterior surface of the sacrum. Besides in this stage, especially with meningeal tumors, circumscribed sensitiveness of the spine, or perhaps a deformity, will rarely be so marked as to render it likely that the pains and paralyses would be ascribed to some vertebral or intravertebral cause. But when in the further course symptoms of compression of the cord appear, first on one side and then on both sides, it will be certain at least that we have to deal with a gradually extending focus of disease upon or within the spinal column; and it is in this stage, in cases with typical course, that in recent times the correct diagnosis of a tumor has been repeatedly made, that is to say, in the transition stage from root to cord symptoms. Leaving aside for the present spinal syphilis, which in rare cases may produce similar symptom groups, we have to consider the differentiation from the disease which most frequently causes com-

pression of the cord, *i.e.*, caries of the spine. Above all we must consider here those cases of caries which present no distinct changes in the vertebral column for a long time, during which they cause, however, considerable root and cord symptoms—cases that on the whole are not very rare. Under the head of vertebral tumors, we have discussed the differences between caries and tumor, and have stated our conviction that none of them is of decisive importance. The entire course of the affection seems to be of particular importance in the differential diagnosis; when root and cord symptoms develop at all in spinal caries the whole course is more rapid; the persistence of root symptoms for years before cord symptoms set in is rather in favor of the diagnosis of tumor. The termination in spontaneous recovery would permit the exclusion of a tumor; very marked deformities of the spine are more likely in caries. The diagnosis of a meningeal tumor becomes almost absolutely certain in such cases with typical course when there are tumors elsewhere; the writer has succeeded in diagnosing two similar cases.

The diagnosis of a meningeal tumor, therefore, is very probable when in a concrete case the symptoms of marked compression of the cord and its roots at a definite level appear in the order of unilateral root lesion, unilateral and then bilateral cord lesion; and when they have developed gradually in the course of one or more years, so that the root symptoms often have remained isolated for years before cord symptoms manifested themselves. This diagnosis, however, can be made with any prospect of being correct only when root and cord symptoms are clearly marked.

That tumors of the cauda equina, as opposed to the other intravertebral neoplasms, usually begin with bilateral symptoms has been stated above. Mention has also been made of the differential factors between vertebral and intravertebral, and of the latter between extradural, intradural, and true cord tumors.

Hypertrophic cervical pachymeningitis always affects the spinal meninges on both sides. As it extends at once to several portions of the cord, it is associated with widespread paralyses, atrophies, and anæsthesiæ due to pure root lesions, without material implication of the cord. This is impossible in cases of meningeal tumor having a typical course, because in these the transition of the tumor along with the symptoms from one side to the other must take place through and across the cord. Moreover, pachymeningitis not rarely ends in recovery.

Multiple tumors of the meninges might be recognized when, for instance, one of them has already produced definite symptoms of a root and cord lesion, and then root symptoms of an altogether differ-

ent region are superadded. When the tumors are close together, however, the symptoms become mixed; possibly, too, it is only the combination of several that presents the phenomena of a transverse lesion at a definite level. Multiple tumors of the spinal meninges are not rarely associated with brain tumors; in that event the cerebral symptoms may mask those of the cord altogether.

When we have made the diagnosis of a tumor of the spinal meninges, the second task is to determine exactly the level of the tumor. According to Horsley, the cervical enlargement, the borders between the dorsal and cervical cord, and the end of the dorsal cord, form the points of predilection for the development of tumors of the meninges, because at those places the latter leave more room. We have above said something about the symptoms of tumors of the upper cervical cord, of the cervical enlargement, of the dorsal cord, of the lumbar enlargement, and of the cauda equina, but rather in a general way; yet this is not sufficient for our present demands for a segmental diagnosis with a view to operation; for this purpose it is necessary to locate the level of the roots and segments in which the tumor has its seat with perfect accuracy. Such localization is based upon our present knowledge regarding the functions of the several segments and their roots, for the two do not differ from each other. With reference to this subject the reader should consult the section on injuries of the spine, where the functions of each single segment are tabulated after Allen Starr and Edinger (p. 577). All that is necessary in a concrete case is to determine accurately which muscles are paralyzed and which cutaneous regions are anæsthetic, and then to decide by the aid of the table to what level the lesion must reach.

An accurate segmental diagnosis is almost invariably possible only when the lesion has implicated the cord, and not until the cord lesion has become nearly complete. We have seen above that the root lesions generally manifest themselves solely by irritative symptoms, and that these are too vague for a segmental diagnosis. Of greater value are the symptoms of lost radicular conduction, especially paralyses and atrophies of the muscles; the more so when the case has been closely observed from its beginning, and when to the root symptoms are superadded those of interrupted conduction of the cord, and both agree as to the level of the segment. But not until the interrupted conduction of the cord has become approximately complete do the anæsthesiæ and paralyses show themselves sufficiently marked to enable us to deduce the level of the lesion from their extent. The upper end of the anæsthesia then at once indicates to us the upper limit of the cord lesion, and in like manner the extent of the muscular paral-

ysis enables us to determine directly by the aid of our table to what height the affection of the cord reaches.

As a rule we are able to determine only the upper limit of the meningeal tumor. This is sufficient for practical purposes, and it is even a matter of special importance in every case carefully to trace the highest segmental symptoms in the morbid picture, since experience has taught us that in many instances the location of the tumor has been diagnosed too low. At the present time we can guard against this error by Sherrington's law and by the rules applying to segment and root lesions enumerated above. The impossibility, in many cases at least, of saying anything positive about the inferior limit of the tumors is due to the fact that in severe compression all functions below it are paralyzed, and that therefore it is immaterial how far down a tumor may extend from this point of greatest compression. This is especially the case in the dorsal cord, where the anæsthesia is the same below as at the point of compression, for atrophy and electrical disturbances cannot be demonstrated in separate intercostal muscles. We are more favorably placed with reference to tumors at the cervical and lumbar enlargement. In these cases all the muscles whose nerve roots emerge directly at the level of the tumor will show flaccid paralysis, atrophy, and the reaction of degeneration, while the muscles innervated from below the compression are affected with a spastic paresis. In favorable cases, therefore, we may form a positive opinion regarding the entire longitudinal extent of the neoplasm; but we may be hampered by three facts: first, the roots often resist the tumors for a long time; second, when the muscular paralysis is caused by the cord lesion at the point of compression, a distinct reaction of degeneration may be absent; third, pains present even in definite regions below the compression do not always prove that the tumor extends downwards to a corresponding level, for such pains may also be projected eccentrically from implicated central sensory pathways situated at a higher point.

The determination of the highest symptoms to be localized, however, is usually easy and practically this is, as a rule, sufficient. Hence typical cases we have the following facts for the diagnosis: We have a total paraplegia usually spastic, but which high up is perhaps atrophic and flaccid, and a total anæsthesia to a certain limit; above this point some partial symptoms of a cord and especially of a root lesion may be present, and may indicate how far the tumor extends upwards above the place of total compression.

In our previous remarks we have assigned a subordinate importance, with reference to the segmental diagnosis of an intravertebral

tumor, to the root symptoms, especially the pains. In this respect we must make some reservation particularly in tumors of the meninges. For if in a special case we find at a certain level the symptoms of a total transverse lesion, the pains, and especially also zones of hyperæsthesia occurring beyond the limits of the interrupted conduction, may be of great diagnostic importance, since they may point to an involvement of the roots above the total transverse lesion, and constitute besides the highest segmental symptoms, the determination of which we are most interested in. With the further upward growth of the tumor they will likewise extend higher and higher, while anæsthesia has already set in in the regions which were formerly painful by reason of the destruction of the roots adjoining the point of greatest development of the tumor. If we find, for instance, in a certain case total anæsthesia as far as the region of the fifth dorsal segment, and hyperæsthesia and pains in the fourth, these pains can no longer be produced by an affection of the fourth roots, because complete anæsthesia in the region of the fifth indicates total destruction of the fourth segment as well, but they point to an affection of the third or even the second pair of dorsal roots. The cutaneous hyperæsthesia is of greater diagnostic value than the pains alone, for it cannot occur below the point of compression with eccentrically projected pains, but always points to a direct lesion of the respective roots. The zones of hyperæsthesia, according to Allen Starr, take the same form on the extremities as the cutaneous regions of the several roots which they cover; on the upper extremities, for instance, they constitute small strips from the shoulder to the fingers, and require careful search on account of their narrowness.

Individual variations may also occur in the relations of the several spinal roots, especially those for the muscles. Thus Eisler and Paterson have demonstrated that the fibres for the peroneal muscles are usually given off from the fourth, more rarely from the fifth lumbar root. Paterson speaks of the prefixed and postfixed type, and careful personal observations have shown us that while in most cases the fibres for the small hand muscles are derived from the first dorsal and the eighth cervical segments, sometimes the seventh cervical segment takes part in the innervation.

It is obvious how important to a segmental diagnosis is the demonstration of a circumscribed sensitiveness of the spine occurring with movements, particularly coughing, or during percussion; this finding will become still more valuable when it agrees with the conclusions drawn as to the level of the tumor from the other examination. In such cases, of course, if an operation is resorted to, the

trepanation will be made at the sensitive spot. When there is no local sensitiveness of the spine, we must select for the operation that part of the column or that spinous process which lies opposite the upper limit of the tumor as indicated by the symptoms. But we must remember that the tip of a spinous process does not correspond with the region of the spinal root bearing the same number, but extends farther down (see page 579). We would especially recall to the reader also the facts of Sherrington's law. If we find, for instance, in a case of tumor the upper limit of the anæsthesia in a cutaneous region innervated mainly by the seventh dorsal root, the sixth dorsal segment, according to Sherrington, must also be destroyed. The origin of the sixth root, however, lies opposite the fourth spinous process, where the upper limit of the tumor must be sought unless irritative symptoms indicate a still higher segment. Should trepanation be undertaken at the limit of the anæsthesia, as an unsophisticated observer might be inclined to do, the opening would be three or four spinous processes too far down, and the tumor would possibly be out of reach.

Not much can be said as to the possibility of diagnosing the nature of a cord tumor. If the neoplasm is metastatic its nature may occasionally be positively determined. If the patient is affected with tuberculosis and the morbid picture indicates multiple tumors situated in the cord the diagnosis of tubercle is reasonably certain. Otherwise sarcoma and fibrosarcoma are most frequent in the meninges, glioma in the cord. Coincident symptoms of a brain tumor would render multiple sarcomatosis of the pia and arachnoid probable.

Prognosis.

The prognosis of a tumor of the cord and its meninges is naturally very grave. Recovery is hardly possible, though the process may be arrested; for instance, a tubercle or a cysticercus may become calcified. Otherwise a tumor according to its nature will steadily progress until it is no longer compatible with life. The prognosis can be, and actually has been, materially improved by the possibility of an operative removal of the neoplasm. This, however, applies solely to tumors of the meninges; those involving the substance of the cord would hardly be subjected to an operation, even if this diagnosis had been made.

Treatment.

Modern medicine may indeed be proud that it has succeeded in making the diagnosis of a tumor of the cord and its seat sufficiently

certain, and in removing the correctly diagnosticated tumor without unduly endangering life and with a prospect of cure or, at least, arrest of the morbid process. This satisfactory result is to be ascribed to the coöperation of the clinician with the surgeon; it is rare to find both qualifications together with those of an eminent physiologist combined in one person as in that of Victor Horsley. The only tumors suitable to an operation are in general those of the meninges, but it is of interest that tumors of the meninges largely exceed in number all the rest.

Prognostically favorable conditions for the surgical treatment of a meningeal tumor are due to (1) the anatomical relations of this affection; (2) the special pathologico-anatomical nature of the tumors; and (3) the influence of the tumor upon the spinal cord proper.

With reference to (1) it should be noted that the space in which a meningeal tumor can develop is small and readily inspected. Most of these neoplasms form on one side of the cord or on its posterior surface so that after removal of the vertebral arches and opening of the dura they come at once into view; but even a small tumor situated on the anterior surface of the cord would be likely to be made visible and accessible by the sacrifice of at most one or two dorsal roots—a sacrifice that will hardly be manifest clinically.

With reference to (2) is to be noted that we have to deal with primary and in many cases isolated new growths which do not tend to form metastases. Moreover, these tumors are usually small—in fact they must be so as long as they do not destroy the bone—and they are generally only loosely adherent, so that they can be readily and completely removed from their matrix. The favorable condition inherent in small size, it is true, exists chiefly in intradural tumors; extradurally we find flat tumors extending over a large portion of the longitudinal axis, whose complete removal would necessitate too great a resection of the spinal column. But aside from such cases we need only look at the illustrations published by Gowers in his text-book of tumors of the spinal cord to recognize at once that most of these cases would have been very favorable for an operation. The assertion of Horsley, however, which Allen Starr also repeats, that *most* cases of spinal tumor offer favorable opportunities for an operation, goes too far.

In (3), the effect of the tumor upon the cord, we find probably the most favorable circumstance as regards the chances of an operation for a spinal tumor. The compression of a slowly growing tumor does not change the true structure of the cord in many cases; in some it merely crowds all the elements of the cross section into a narrower space and in others an œdema of the cord results. In either of the

last-named instances the function of the cord may be lost during the persistence of the compression; but as the structure is preserved, when the pressure ceases, even after years, the normal function may be restored. In many cases, therefore, we may wait quietly until the symptom complex has advanced to complete transverse interruption of conduction, so that an accurate general and segmental diagnosis can be made; and in cases which do not come under observation until the compression is of long standing there is still hope that the function will return after the pressure is relieved. It does not matter if some single roots have in the mean time been definitively destroyed.

In order to make the above-mentioned favorable conditions practically available, it is of course necessary that in every case the diagnosis of a spinal tumor and its seat be reasonably certain. As we have seen, the diagnosis of an obscure spinal tumor is rendered probable by the course and grouping of the symptoms presented by the cord, and this diagnosis becomes positive when in the other parts of the body neoplasms are discovered which are similar in their nature to those occurring in the spinal meninges. Nor is it necessary that these be true metastases—for in that event the certainty of the diagnosis would be counteracted by the bad prognosis, although one of Kümmel's cases shows that even then good results may occasionally be obtained—there may be simply a simultaneous development of several tumors in different regions. Nothing more need be said here about the segmental diagnosis, in view of the full discussion of the subject under the head of injuries of the spinal cord. When the above rules are followed an erroneous diagnosis will be rare indeed, and in carefully observed cases the segmental diagnosis likewise will be so positive as to almost force us to decide upon an operation. For all these reasons it is not surprising that even years ago men of experience became convinced that the surgical removal of a spinal tumor in cases in which the diagnosis was positive was most desirable, and that nothing more was needed than greater technical skill and reliable asepsis to furnish successful results in favorable cases. Leyden was probably the first to take this bold plan into consideration, for he said in 1874, that the only means [of cure] would be extirpation after trepanation of the spine, and that such a procedure would be no less feasible than in spinal fractures. Leyden considered these matters not only from a purely theoretical standpoint, but in a correctly diagnosed case of obscure spinal tumor he also weighed the chances of an operation. Erb also said in 1878: "When the diagnosis and the localization of a tumor are absolutely certain, trepanation of the spine might be considered; . . . a successful result is by no means impossible, especially in a case

of tumor outside the dura, upon its posterior surface." Gowers said in 1886: Modern methods render the opening of the spinal canal much less dangerous than it was formerly, and the removal of a tumor of the spinal meninges must be associated with less immediate dangers than that of a cerebral tumor." While Leyden and Erb were doomed to wait long for the realization of their hope, that of Gowers was soon fulfilled in the famous case observed by him and Horsley, and which was operated upon by the latter (1887).

1. This was the case of a man aged forty-two, who began to suffer in 1884 from intercostal pains under the left scapula, which continued with varying intensity until February, 1887. Later there followed weakness and loss of sensibility of both lower extremities, then retention of urine. In June, 1887, there were paraplegia and total anæsthesia as far as the ensiform process (according to Head the region of the sixth and seventh dorsal roots); at this level and somewhat above it severe radiating pains were found, hence corresponding to the sixth and fifth dorsal roots; a short time prior to operation sensation was abolished in the fifth intercostal space (sixth dorsal segment) on both sides, and was uncertain on the left side in the fourth intercostal space, the main region of the fifth dorsal root. Pains were present in the distribution of the fifth and sixth dorsal segments. Horsley, according to the views then prevailing, correctly referred the highest segmental symptoms of his case to the level of the fifth dorsal segment and operated so as to strike the upper margin of this segment. He removed the fourth, fifth, and sixth dorsal arches, thus exposing the fifth dorsal root in its entire length. He failed to find the tumor there, but found its lower limit at the origin of the fourth dorsal root after removal of the second and third dorsal arches in addition, and the upper limit which extended to the third dorsal root was not reached until the first arch was also taken away. The tumor was a fibromyxoma the size of a filbert and did not involve the cord. Ten days after the operation the right leg could be moved, after six weeks the left, and finally the patient could walk three miles and passed his urine and fæces without difficulty. Horsley operated too low because he was not acquainted with Sherrington's law as to the anastomosis of the several roots, and also because he confused root regions and intercostal spaces with each other. Operating at the present day he would have struck the tumor at once without difficulty. In the presence of complete anæsthesia in the distribution of the sixth dorsal root on both sides and with considerable dysæsthesia in the fifth segment, the latter must have been totally and the fourth or its root materially injured; the pains in the fifth dorsal root region then could hardly be still referred to a lesion of the largely involved fourth root, but would point to an implication of the third.

The sensation produced by this notable case of Horsley and Gowers was a lasting one. One of its effects above all was that clinicians and

physiologists endeavored more and more to place the segmental diagnosis on a reliable foundation. What has been done in recent years in this respect is stated at length in the diagnostic part of this section and in that on injuries of the spinal cord; here we need but mention the names of Sherrington, Head, Mackenzie, and Thorburn. The rest of the clinical symptoms of cord tumors, the condition of the cord and its functions, particularly the tendon reflexes below the point of compression, the relation of the cord to the tumor, and the pathological anatomy of the neoplasms are studied with increasing care. Horsley himself set a good example, and not the least service was rendered by clinical observations and pathologico-anatomical examinations, especially in the cases subjected to operation (Bruns, Allen Starr). Owing to this enlargement of our clinical and particularly our diagnostic knowledge the number of tumors thus far operated upon has become considerable, and in view of the importance of the subject it is worth while to pass them briefly in review. According to the literature at our disposal the number comprises twenty cases, including that of Horsley-Gowers.

2. Horsley (*British Medical Journal*, 1890, II.). Complete paraplegia and painful attacks for six months. Trepanation. The tumor covered the dura for a distance of four inches. Death from shock.

3. Roy, 1890 (Chipault). Paraplegia, girdle pains, etc. Removal of the arches of the lowest four dorsal vertebræ; intradural tumor. Rapid improvement after its removal. The sensibility returned, the paralysis of the sphincters disappeared, ultimately the patient could walk with the aid of a cane.

4. Laquer and Rehn (*Neurologisches Centralblatt*, 1891, p. 193). The diagnosis was tumor of the cauda equina. The principal symptoms were pains, slight vesical disturbance, atrophy of the quadriceps muscles, and absence of the patellar reflexes. According to these symptoms the diagnosis was perhaps not quite certain. The operation showed a cavernous lymphangioma in the sacral canal. Marked improvement.

5. Fenger, 1890 (Chipault). One year previously painful attacks in the lumbar region. Three months later, weakness of the right leg, girdle sensation midway between umbilicus and symphysis. Subsequently weakness followed in the left leg. Then lancinating pains in the legs, constipation, and paralysis of the bladder. Below the fourth rib complete loss of the temperature sense and diminution of the pressure sense. Reflexes increased in the lower extremities. Operation. The arches of the fourth and fifth dorsal vertebræ were removed and the dura was incised. The cord was distended by a spindle-celled sarcoma situated in the posterior columns, which was enucleated. Complete paraplegia after operation. Death from sepsis on the fourth day.

6. Ramson and Anderson (*British Medical Journal*, 1891, II., 1144). Lumbar pains, radiating into the legs. After four months

they ceased and returned later. Then followed paraplegia, retention of urine, anæsthesia of the legs, paralysis of the rectum, and bed-sores. Patellar reflexes absent. Operation. Removal of the arches of the eleventh and twelfth dorsal, first and second lumbar vertebræ. The dura was opened but nothing found. Death after three days. Autopsy—in the dorsal muscles two echinococcus cysts, another one under the arches of the tenth dorsal vertebra. (In this case the operation was too low, a mistake which would not be made now.)

7. Pesearolo (Transactions of the Tenth International Congress, Berlin, IV., 59). Paraplegia with incomplete vesical and rectal paralysis present twelve years, anæsthesia reached to the fifth intercostal space. Previous to the paralysis neuralgias existed in the third to fifth intercostal spaces. Tendon reflexes exaggerated. Diagnosis, tumor between the second and fifth dorsal vertebræ. The arches of these vertebræ were removed and a tumor situated to the left of the cord was found and extirpated. The cord was much thinned. As was to be expected in view of the long duration of the paralysis, there was no improvement except in the reflexes.

8 and 9 are cases by Lichtheim and Mikulicz, which have thus far been only very briefly reported (*Deutsche medicinische Wochenschrift*, 1891, p. 1386). These were two cases of compression of the cord by a psammoma of the dura mater. Both tumors were removed by Mikulicz. In the first case (operation on June 19th, 1890) the tumor was at the level of the ninth thoracic vertebra. The patient died of sepsis two days after operation. In the second case, operated upon September 25th, 1890, the tumor was at the level of the fourth thoracic vertebra. After recovery from the operation the interruption of conduction, which had been total about six months, so diminished that the patient could walk without the aid of a cane. Some disturbances of sensibility persisted in the right leg with slight ataxia on movement, also an anæsthetic semicircle at the level of the fourth rib around the right half of the thorax, due to a lesion of one [?] posterior root. (Reported thirteen months after operation.)

10. Ramson and Thompson (*British Medical Journal*, 1894, I., p. 395). Locomotive engineer, aged 50 years. Trembling, weakness, stiffness, and pains in the legs. Patellar reflexes increased. Subsequently ankle clonus, anæsthesia, inability to walk. Sphincters normal. Girdle sensation. The left leg was weaker than the right; both legs were partially anæsthetic. Sensitiveness along the eighth and ninth dorsal vertebræ. Diagnosis, tumor. The arches of the fifth to the ninth dorsal vertebræ were removed and a tumor was found at the level of the eighth dorsal root (round-celled sarcoma). Death after three days.

11. Bruns and Kredel (*Neurologisches Centralblatt*, 1894, No. 3 and 1895, No. 7; *Archiv für Psychiatrie und Nervenkrankheiten*, Bd. XXVIII., p. 97). A young woman began to be affected in the summer of 1890 with pains in the sacrum and the lower lumbar spine, radiating into the lumbar distribution first of both legs, then only into that of the right leg, and later involving also the distribution of the sacral nerves. The painful period lasted eighteen months, with long remissions. Early in 1892 paralysis occurred in the distribution of the right peroneal and tibial nerves (foot immovable), without

marked atrophy and without qualitative electrical disturbances until autumn of the same year. During this time no distinct or even partial anæsthesia could be discovered anywhere. The pains continued. A small fibrosarcoma on the right ear was extirpated. In the summer of 1892 the first vesical disturbances set in. In August of that year paraplegia occurred rather suddenly, after very indefinite symptoms of a right unilateral lesion. In September the anæsthesia extended into the first lumbar region, with paralysis, atrophy, and reaction of degeneration of all leg muscles, at first predominating in the lumbar distribution. Œdema; complete vesical and rectal paralysis. Diagnosis, tumor (sarcoma) adjoining the right side of the cord, centering at the level of the fourth or fifth lumbar root, but extending as high as the first. Compression of the cord. While a caudal tumor was excluded, the possibility of its presence was borne in mind during the operation. We did not know at that time that in total anæsthesia in the first lumbar distribution the eleventh dorsal root must be involved in the destruction. On October 22d, 1892, the ninth to twelfth dorsal and the first lumbar arches were removed. The cord appeared very thick and did not pulsate, but nothing else was found. The patient lived fourteen months longer, relatively free from pain in the second third of this period. Few changes occurred in the symptoms, only the pains and the limit of the anæsthesia gradually rose higher. Death in December, 1893. At the autopsy the lumbar cord and the second and third lowest dorsal segments were found changed to a compact tumor; in the neighborhood of the ninth dorsal root thick tumor nodules lay upon the posterior columns and in the arachnoidal tissue surrounded the entire cord; at the level of the eighth segment especially the posterior and anterior roots were transformed into tumor nodules the last remnants of which could be followed as far as the fourth dorsal segment. Microscopical examination showed the neoplasm to be a multiple sarcoma of the meninges, which had involved the cord. In this case, despite the correct diagnosis, success was not attainable.

12. Sänger and Krause (*Münchner medicinische Wochenschrift*, 1894, No. 22). Tobacco worker, aged 42 years. Violent pains on the left side of the chest present for nine months. Admitted to the Altona Hospital on March 6th, 1894. Of late, pains had appeared also on the right side of the chest. Stiffness and coldness of the legs present two weeks; walking had become impossible three days before admission. April 10th, complete paraplegia with partial analgesia, painful girdle sensation. Pain on pressure from the seventh to the tenth thoracic vertebra. The anæsthesia on the back extended on the left to the seventh, on the right to the tenth thoracic vertebra. Tumor diagnosed between the fifth and seventh dorsal vertebræ. Operation April 16th; the arches were removed from the fourth to the seventh dorsal vertebra and the dura was opened. A small tumor was found on the left side in the region of the sixth thoracic vertebra. The cord was flattened from the left. Death four days after operation. Subdural hemorrhage in the brain, extending thence into the cord.

13. Bruns and Lindemann (*Neurologisches Centralblatt*, 1894, p. 359, and 1895, No. 3; *Archiv für Psychiatrie und Nervenkrankheiten*, Bd. 28). A young man, operated upon for tumor three times

during the last six months (teratoma on the vena cava in the abdominal cavity, sarcoma in the supraclavicular fossa, and one on the right testicle). Soon after the last operation on April 9th, 1894, pains were felt in the left cutaneous distribution of the third dorsal root (according to Head) above the nipple; no pain at first on the right side. Beginning April 12th gradual development of paraplegia, first paralysis, then anæsthesia, advancing from below upwards; followed by vesical and rectal paralysis, while the pains in the left third intercostal space continued. When the paraplegia was complete (April 23d) both patellar reflexes were lost, but the plantar reflexes were preserved. At that date (a day before operation) total anæsthesia reached to the fourth dorsal segment and sensation was very uncertain in the third. Pains on both sides formed a belt around the chest in the third dorsal segment and were present also in the distribution of the second dorsal root (ulnar side of the arms), but no paralysis and anæsthesia anywhere in the arms. Total paraplegia, and paralysis of the bladder and rectum. As there was total anæsthesia in the fourth dorsal segment the lesion of the third root was certain to be profound and to implicate considerably also the second dorsal segment or its roots. The latter was indicated besides by the blunted sensation in the third dorsal segment and especially by the pains in the distribution of the second dorsal root in the arms. Pains had been present from the beginning in the distribution of the third root and this was therefore probably directly involved. Moreover, corresponding to these roots the space between the first and second dorsal spinous processes and the latter itself were markedly painful on pressure. While the diagnosis of a tumor and its seat was very easy, the operation was undertaken only in compliance with the urgent request of the patient and his wife, since we had evidently to deal with a malignant metastatic neoplasm. Resection of the first and second dorsal arches was performed. At the upper margin of the third dorsal root segment was a flat, very vascular, extradural tumor (sarcoma of the extradural adipose tissue), which covered the entire posterior surface of the cord. After removal of the third and fourth dorsal arches this tumor could be followed as far as the sixth dorsal segment. It was extirpated as far as feasible. The dura was not opened. The patient died of shock the same afternoon. The autopsy showed that some small remnants of the tumor had been left above and below. Macroscopic and microscopic examination of the cord revealed total softening of the third and fourth dorsal segments, diffuse myelitis at the level of the second, and scattered foci of degeneration with many recent hemorrhages; the first dorsal segment was nearly normal. Some diffuse myelitis was also present in the fifth and sixth dorsal segments. There was nothing abnormal in the lumbar cord except degeneration of the lateral pyramidal tracts. In view of the findings in the third and fourth dorsal segments functional restoration could hardly have been expected.

14 and 15 are cases by Abbe, of which we have seen only brief abstracts by Allen Starr (*Medical Record*, 1889 and 1890). The first case was that of an extradural tumor extending from the eighth to the tenth dorsal segment. The tumor was extirpated; the patient recovered from the operation and the paralysis improved.

In the other case an extradural sarcoma was removed from the eighth and ninth dorsal segments. The patient died on the ninth day after the operation.

16. Starr and McCosh (*American Journal of the Medical Sciences*, June, 1895). Onset with rather vague pains in the abdomen and in the right shoulder. Gradual development of a markedly spastic paralysis of the legs and of the abdominal muscles, clonic reflexes, anæsthesia up to the distribution of the seventh dorsal root with superimposed zones of hyperæsthesia; pains round about the abdomen, likewise in the distribution of the seventh dorsal root. No vesical disturbances. The extradural sarcoma was found directly under the arch of the fifth vetrebra; it surrounded the cord almost completely, was flat throughout and very vascular. It was entirely removed. No improvement followed; the patient died about two weeks later of marasmus.

17. The same operators (*ibidem*). A few weeks previously the patient had been operated upon for a lipoma of the right knee-joint. Then she began to suffer severe pains in the distribution of the lumbar plexus, *i.e.*, particularly at the anterior surface of the thighs. Anæsthesia was present in the same region; sensation was good in the sacral distribution. Weakness of the legs with increased reflexes and spastic symptoms. Vesical disturbances. Diagnosis, lipoma of the lumbar cord. At the operation such a tumor was found extradurally, corresponding to the upper part of the lumbar cord, and was removed; an additional small tumor was situated above it. Only very slight improvement followed; the patient ultimately died of caries of the upper dorsal spine.

18. Starr and McBurney (*ibidem*). Tubercle on the outer surface of the dura, corresponding to the upper and middle portions of the lumbar enlargement. The symptoms of spinal caries had been present for a long time before cord symptoms appeared. The extent of the anæsthesia pointed on the right side to implication of the second, on the left to that of the third lumbar segment. Spastic paralysis of the legs; vesical disturbances. The tubercle was removed. Great improvement of all symptoms to the end of the fourth week; then followed acute gastritis and renewed exacerbation of the symptoms. A second operation disclosed additional tuberculous masses which could not be removed. The patient died a few months later.

19. Mitchell Clarke (*Brain*, 1895). Flaccid paralysis, atrophy, and electrical disturbances in both hands and forearms; paresis of the muscles of the upper arm and shoulder. Spastic paralysis of the legs, with patellar and Achilles clonus. Vesical and rectal paralysis. Sensation abolished on both forearms, behind on the upper arm, on the trunk from the second rib to the inguinal fold; sensation present on the thighs, but the legs were anæsthetic. Hyperæsthesia from the third to the fourth rib. No severe pains. Diagnosis, tumor in the lower portion of the cervical enlargement. At this point the operation disclosed an extradural endothelioma, which was removed. Profuse hemorrhage. Death some hours after operation.

20. Kümmel (*Archiv für klinische Chirurgie*, Bd. 50, 1895). In 1893 operation for a sacral sarcoma. In March, 1894, gradual development of a spastic paraplegia. Hyperæsthetic zones above the third

thoracic vertebra. Diagnosis metastatic sarcoma. This, as shown by the operation, had sprung from the third and fourth vertebral bodies but could not be removed. Marked improvement. The patient could walk well, but the bladder remained paralyzed. This was really a tumor of the spinal column.

On reviewing this considerable number of operations for cord tumor we cannot fail to recognize that the experience gained by them will certainly justify such surgical treatment in similar cases. In the first place, the diagnosis of the tumor was correct in every instance. While the objection may be made that as a rule operative cases with erroneous diagnosis are not likely to be reported, still to the best of our knowledge thus far there has been only one case—to be mentioned hereafter—of erroneous diagnosis which was published with commendable candor; and besides, this can in no way alter the fact that nowadays the correct diagnosis of a cord tumor is feasible in a much larger number of cases than would have been expected not long since. And, what is of equal importance to an operation, in most instances the diagnosis of the level was successful; only in the sixth case above enumerated was the operation too low and the tumor was not found. If Horsley and Gowers in their case had not been so certain of their diagnosis that they were not deterred by their first negative finding but looked higher, they would have met the same fate.

In 18 of the 20 cases the correctly diagnosed tumor was removed during the operation; in one instance it was not found because the opening was made too low; in Bruns' first case (No. 11) because it was a flat sarcoma of the meninges which was not recognized during the operation and besides could not have been removed. In 6 of these 20 cases marked improvement and even recovery was attained, that is, in exactly thirty per cent.; in 2 only very slight and in one temporary improvement followed. In 12 cases death occurred; 9 soon after operation by shock, after-hemorrhage, and sepsis; 3 at a later period by relapse, marasmus, and extension of the tumor, which was not discovered during the operation (No. 11). Thirty per cent. of cures or improvement is quite a favorable result in a disease which unless relieved by operation inevitably ends in a most painful death. Besides, death from sepsis will probably be avoidable and perhaps also cases of fatal shock from hemorrhage or prolonged operation will lessen in number. The most important point in improving the prognosis of this operation is probably still greater certainty in the diagnosis, which will enable us to operate as early as possible, and this will doubtless be attained, judging from the experience of recent years. In short, we are not only justified but morally obliged in every

case of correctly diagnosed tumor of the cord to advise the patient to submit to an operation.

Thus far we have intentionally pointed out everything in favor of an operation for a cord tumor. It now becomes our duty to paint also the shadows of the picture presented, for the matter is by no means so favorable as was assumed after the first brilliant result. This is proved by additional experience and by a perusal of the cases above enumerated, and we are forced to differ decidedly from Horsley and Allen Starr, who assert that most tumors of the cord may be operated upon with a prospect of success. Many things concur in nullifying the final practical result even in carefully observed cases. The first of these are rather surgical in their nature. The operation is always a dangerous one; it is penetrating and prolonged; the loss of blood is very great during the mere division of the long back muscles; the hemorrhage from the cord and tumor is often arrested with difficulty, especially in flat extradural sarcoma (cases 13 and 19). Moreover, as stated before, according to the more recent observation of Chipault, the opening of the dura and the escape of cerebrospinal fluid may directly jeopardize life.

A second factor against the operation is that while, as stated above, in carefully observed and typical cases the diagnosis is often successfully made, it is always a most difficult one. In cases otherwise favorable the diagnosis, as we have said, may become positive when tumors are found in other parts of the body. Of course with true metastatic tumors the greater certainty of the diagnosis is counteracted by the worse prognosis, and the writer must confess that in his second case (No. 13 in the list) he hesitated long and decided to operate only in compliance with the urgent request of the patient and his wife. Since then Kümmel's case (No. 20) has shown what good results may be obtained even in metastatic cases, and therefore the operation must be regarded as justifiable.

In the absence of tumors in other organs the diagnosis of a cord tumor may be a very probable one but will hardly ever be quite certain; for the case may prove to be one of caries of the spine, of simple myelitis, or an inoperable tumor of the vertebral column. The case above referred to, of an operation under an erroneous diagnosis of a cord tumor, was that of F. Schultze and Pfeifer and it shows that such a mistake may happen even to experienced observers. The diagnosis was based upon prolonged and rebellious pains located at a definite spot and associated with slight cord symptoms, but no tumor was discovered at the operation or at the autopsy. The case proves that it is wise to defer the diagnosis of a meningeal tumor until the cord symptoms have become marked; root symptoms and

especially pains, no matter how persistent and how strictly localized, do not suffice for making this diagnosis.

The segmental diagnosis of a tumor is at all events more difficult than that, say, of a trauma. The latter in general produces much simpler conditions; usually it abolishes function, at least temporarily, at a definite transverse level. A tumor may never do so; it may cause merely a partial lesion which does not admit of a positive diagnosis of the level; or else the symptoms of interrupted conduction at a certain point are produced by the combined effect of several tumors each of which by itself causes only a partial lesion. For this reason a positive segmental diagnosis will be impossible. But even when the general and segmental diagnosis of the tumor is absolutely correct we may meet with an unexpected surprise during the operation, as is often the case with brain tumors. This happened, for instance, with No. 11 in the list, in which the correctly diagnosticated tumor was a flat sarcoma of the meninges that could not be extirpated or even recognized during the operation. Besides, it was a case of multiple tumors—a condition only rarely discoverable; if such a diagnosis can be made, which is most likely with simultaneous cord and brain tumors, an operation would of course be out of the question. Complete extirpation is also impossible in flat extradural tumors, as in cases 13 and 19. Their length is often so great that their complete removal would require the sacrifice of too much of the spine; besides, their inferior limit is unfortunately not to be diagnosed, as a rule.

Moreover, although the arrested function of the cord resulting from pressure of a tumor is usually, as we have seen, due to conditions capable of repair, such as œdema and simple compression, still complete softening may set in, as in case 13, so that functional restoration will fail even after the pressure is relieved. Such softening will generally be ischæmic or inflammatory in its nature. It cannot be diagnosed, for a benign œdema in meningeal tumor may produce as rapid a paraplegia as an irreparable softening; a slowly developing, perhaps at first unilateral, then bilateral paralysis would indicate rather a benign cause of the interrupted function.

Finally, the differential diagnosis between a tumor of the cord and one of the meninges is very difficult and usually impossible. When the diagnosis of a cord tumor is positively made an operation will probably be out of the question, since recovery or even improvement is not to be expected and the enucleation of the tumor from the cord could only make the symptoms worse. When the diagnosis between a tumor of the cord and one of the meninges is in doubt, and on

operation a cord tumor is found, which is sharply demarcated and has completely destroyed the cord, the best plan would be to resect the entire portion of the latter. Additional morbid symptoms will not be caused thereby, but a further spread of the tumor and the continual recurrence of pains may be prevented. Of course, owing to the narrowness of the field of operation, the recognition of the limits of a cord tumor will always be difficult.

These are all of the difficulties which in cases of correct diagnosis may destroy our hopes for a favorable result from the operation. But none of them should prevent us from advising an operation in every case in which the diagnosis is well founded. They may, however, cause us not to press the matter too urgently. In these cases as in brain tumors it is the writer's habit, whenever possible, to explain to the patient or his relatives all the chances of the operation and this in not too hopeful a strain, leaving the decision to them. It will be a matter of tact to give our information in such a way as to spare the patient as much as possible. A patient who suffers much will usually decide for an operation.

A few words may be added as to the non-surgical treatment of cord tumors, and this is to be divided into two periods. First, that before operation. This includes above all an antisyphilitic treatment and in view of the frequency of syphilitic diseases of the cord with symptoms resembling those of tumors it should be instituted in all cases in which the possibility of infection or of hereditary syphilis is present. The cases in which it can be absolutely excluded, as in No. 13 of the list, will be rare, and in these no time will be wasted over a course of inunctions. Otherwise, the time devoted to antisyphilitic treatment, which will be begun in the stage in which the diagnosis is uncertain and root symptoms predominate, will be utilized in perfecting the general and segmental diagnosis in every direction by continued careful observation.

The second period of non-surgical treatment covers the time after unsuccessful operations, or the whole course of such cases which cannot be operated upon either because of uncertainty of the diagnosis or because of the refusal of the patient. Its main object is the mitigation of the severe pains, for which purpose morphine and hypnotics should be freely used. Careful general management is self-evident—it comprises the bedding, cleanliness, supervision of the vesical and rectal evacuations, and the prevention of bedsores. The details have been discussed under the head of vertebral tumors.

The writer may venture to make some further remarks, although they encroach upon surgery. We have seen that in order to remove the tumor a large number of vertebral arches must often be sacrificed.

This is of no consequence at the lumbar spine and particularly at the sacrum. At the cervical and upper dorsal spine, however, the maintenance of the head in an erect position is rendered impossible by the removal of several vertebral arches. For this reason it is advisable in every case to try Urban's osteoplastic resection of the vertebral arches, in which the skin, muscles, and bone remain in connection and are turned back to their place after the completion of the operation. The latter of course is thereby made much more difficult and certainly will take longer to perform. Besides, some pieces of bone occasionally become necrotic.

In case No. 13 it was found on microscopical examination of the cord above the softened focus that numerous recent hemorrhages had occurred at this point which was besides no longer sound. As most of the hammering and chiselling during the opening of the spine was done opposite this region, there is no doubt that these hemorrhages were due to the operation. As such hemorrhages are far from harmless it will behoove surgeons to avoid them by employing milder methods of operation.

The trepanation must always be performed upon the arch corresponding to the segment in which the highest symptoms are to be localized. As a rule the tumor will be found at this point; if not, we must go farther down; possibly, however, another higher arch may also be sacrificed.

HÆMATOMYELIA.

Definition.

In this place we shall speak only of the primary hemorrhages into the substance of the spinal cord (spinal apoplexy), which are the result of injury to one or more of the small vessels of the cord. We do not of course take in consideration whether these vessels were previously perfectly healthy or were affected by arteriosclerosis, or whether a congenital (hæmatophilia) or acquired (scorbutus) hemorrhagic diathesis of the individual previously existed. Secondary hemorrhages in cases of tumor, especially glioma, or of diffuse myelitis or poliomyelitis are excluded. A red softening may also occur in thrombosis of the blood-vessels of the spinal cord in the area of these vessels. This disease, the frequent occurrence of which has until lately been underestimated, was formerly looked upon generally as a hemorrhagic form of myelitis. These cases, however, do not belong to hemorrhages proper of the spinal cord.

History.

Our knowledge of spinal apoplexy dates as far back as the first anatomicopathological studies of the spinal cord. For a long time no sharp distinction was made, and cases of hemorrhage in glioma as well as in inflammation and thrombotic softening were grouped together with primary hæmatomyelia. When, however, it was discovered, especially through the researches of Charcot, Hayem, and Koester, that a great number of cases which up to that time had been looked upon as primary hemorrhages, were really secondary hemorrhages originating in true or supposed cases of myelitis, Hayem went so far as to deny the occurrence of primary hemorrhages of the medulla; according to his view, hemorrhage into the cord occurred only as a result of inflammatory processes. This opinion could be entertained only because at that time myelitis was considered of more frequent occurrence than it really is, the softening of the spinal cord, which is not infrequently due to thrombosis, and in which we of course frequently meet with blood extravasation, being looked upon as a true myelitis; and furthermore, the symptoms of reaction which present themselves in the neighborhood of primary blood extravasations were also regarded as evidences of a primary inflammation.

In addition to all these sources of error Hayem neglected entirely the primary traumatic hemorrhages and those occurring in arteriosclerosis, notwithstanding that Lionville and Goldammer had positively demonstrated the presence of the latter. The greater number of later authors, especially Leyden and Erb, have always admitted the fact of primary hemorrhage of the cord, although they may have regarded it as of rare occurrence. As concerns our knowledge of traumatic hæmatomyelia we are particularly indebted to Minor.

Etiology.

A hemorrhage of the spinal cord, analogous to the common form of cerebral apoplexy, is undoubtedly of rare occurrence. As we are not able to assume that the arteriosclerosis, which is so frequently present, stops short at the spinal cord (on the contrary is usually found quite marked in the spinal cord in old age); as furthermore, miliary aneurysms, which are regarded by Charcot and others as of special importance in the genesis of apoplexy, have also been seen in the spinal cord, the infrequency of apoplexy of the spinal cord itself must be attributed to other causes. Gowers believed that the blood pressure was rarely as high in the arteries of the spinal cord, on ac-

count of their tortuosity and length, as in the cerebral arteries. We nevertheless do meet, even if rarely, with hemorrhages of the spinal cord, of sufficient extent to cause distinct symptoms, as the result of a spontaneous rupture of blood-vessels affected by arteriosclerosis; smaller miliary hemorrhages are in all probability not so very rare.

It was formerly supposed that other lesions depending on vascular disease were more rarely met with in the spinal cord than in the brain. This may be true of embolism, but thrombosis occurs much more frequently than was formerly believed, although it certainly is less common than thrombosis of the cerebral blood-vessels. Whether nephritis, which more frequently leads to changes in the heart and blood-vessels in the young, may also be a cause of hemorrhage in the spinal cord, is not yet positively determined. It is very probable that spinal hemorrhage occurs in cases of scurvy and of purpura hæmorrhagica.

The most important cause of a pure uncomplicated and primary hæmatomyelia is undoubtedly traumatism. According to Oppenheim this is the cause of hemorrhage of the spinal cord in nine-tenths of the cases. Cases of severe destruction of the spine and of the spinal cord in which hemorrhage is only an accidental factor need not, of course, be considered here. We are concerned chiefly with those cases of injury to the back which are not of sufficient severity to cause a fracture or dislocation of the vertebræ, but as a result of which hemorrhage has taken place into the tissues of the spinal cord; this occurs in cases of violent blows on the back, of falls on the head, on the buttock, or on the feet, or even of forcible bending of the spine to one or the other side. Quite frequently a bending of the spine and of the cord at about the junction of the cervical and dorsal cord is produced by a fall on the head; the spinal cord is here strained and hemorrhage occurs at the spot which receives the greatest strain, leading to a destruction of the cord without any evident lesion of the spine. A special cause of multiple and extensive hemorrhages came under the writer's observation in a case of tumor of the spinal cord. In this case the hemorrhage was obviously produced by the concussion to which the cord, of course previously diseased, was subjected in the process of opening the spinal canal in order to remove a tumor.

Many minor causes, which it is rather straining a point to class with traumata, have given rise under certain circumstances to hemorrhage of the spinal cord. Among these the most frequent is perhaps the lifting of a heavy weight on the shoulders. Oppenheim mentions other cases, for example, a soldier drilling with a rifle, and a person possessed of considerable strength who tried to break a piece

of wood with his feet. Gowers mentions the occurrence of spinal apoplexy after coitus practised four times at brief intervals, the hemorrhage occurring during the fourth act. It is probable that such slight causes can produce a hemorrhage only in case of disease and brittleness of the blood-vessels or of the hemorrhagic diathesis.

We may furthermore mention that suppression of the menses or of a hemorrhoidal flux is said to have been the cause of spinal apoplexy. From the preponderance of traumatism in the etiology of hemorrhage of the spinal cord it is very natural that it should occur much more frequently in men than in women, and with special frequency during the most robust period of life.

Pathological Anatomy.

Hemorrhage into the tissues of the spinal cord shows a marked preference for the gray matter. In this location it may involve the whole cross section of the anterior and posterior horns or only the anterior horns, or even only one anterior or one posterior horn. Typical cases of the latter are described by Minor. In the white matter the hemorrhage occurs by preference in the posterior columns, and especially in the connective-tissue septa of the latter; in the cervical cord in the sulcus intermediolateralis. Both points of predilection are based on the fact that the gray matter and the above-mentioned septa contain the greater number and also the largest blood-vessels, and that the tissue of the gray matter is looser than that of the white matter. The extent of the hemorrhage laterally is usually limited, but it is for that reason greater in a longitudinal direction. In extreme cases the hemorrhage may involve the whole length of the gray anterior and posterior columns (the so-called tubular hemorrhage), but usually it extends over only a few segments. As a general rule, a hemorrhage in the cervical or lumbar enlargement takes place more readily than one in the dorsal region.

If we look at a spinal cord into which a hemorrhage has recently taken place, the blood clot may often be visible through the surrounding white matter, as a mass of a bluish-red color; in severe cases of so-called tubular hemorrhage the whole spinal cord may form only a thin-walled sac surrounding the blood clot. The cord is then somewhat increased in diameter. On cross section the blood clot is seen to be made up of red blood corpuscles and the remains of nerve tissue. If we examine recent cross sections microscopically, we notice that the blood clot does not stand out in relief from the healthy tissue with perfectly well-defined borders, but in consequence of the effusion as well as of the accompanying œdema, we

find more or less destruction of the nerve substance. This destruction was formerly designated as secondary myelitis, which is not altogether correct. If the clot is somewhat older, we shall find the blood corpuscles in all stages of disintegration and destruction; free coloring matter of the blood will be found abundantly, and along with it broken portions of the cord, as well as free myelin and granular cells. The granular cells are found more especially in the immediate neighborhood of the clot, but there is also seen here a hyperplasia of the neuroglia, and especially an increase in the number of blood-vessels. In the case of very small clots cicatricial tissue may be formed, whose origin in a hemorrhage may be recognized only by its richness in pigment granules and especially in hæmatoidin. More frequently, especially after a large hemorrhage, the blood may be dried up into a crumbling mass, or else it becomes more and more fluid. In a very few cases only does absorption progress so far as to leave behind simply a serous cyst, such as we so frequently find after cerebral hemorrhage; this cyst is then enclosed in a capsule of more or less firm connective tissue. The unimportant changes in the neighborhood of the clot itself usually disappear altogether. Quite frequently we find long cracks or hollow tubes in the spinal cord after a hæmatomyelia, especially in traumatic cases; they may gradually extend upwards to a great distance from the blood clot. These are found most frequently at the root of the posterior horns. Later on they may be filled with serum; their walls are formed by an increased growth of the glia, which is usually completely filled with granular cells. It was formerly believed that these were always caused by hemorrhages, but we now know that they may also be produced by a long-continued necrosis due to traumatism, after the necrotic portion has been absorbed. The proof of this is furnished by those cases in which the necrotic plugs are still to be seen loosely filling the tube. These tubes and fissures remind one very much of syringomyelia, and it has been contended that the latter affection may so arise. Secondary ascending and descending degeneration may be met with when the white matter is extensively invaded. It goes without saying that the peripheral nerves and muscles dependent on the destroyed segments also become degenerated.

In recent cases, in which we find the clot composed wholly of red blood corpuscles, the diagnosis is probably easy. Later on, however, when the blood corpuscles commence to disintegrate, when granular cells are found in the clot, when the neighboring parts are plainly breaking down, and when hyperplasia of the glia and blood-vessels has appeared, the differentiation from myelitis may be very difficult. In its more minute histological appearances a "myelitic"

clot is hardly distinguishable from a contusion and hemorrhage of the cord. Much more difficult still may be the differential diagnosis from thrombotic softening; and even in glioma with hemorrhage the demonstration of a neoplasm, which occasionally occupies only a narrow border around the clot, is not always an easy matter; frequently, therefore, we have to rely upon a history of the appearance of the disease immediately after a traumatism in order to establish the diagnosis.

Symptoms and Course.

The symptoms of hæmatomyelia in the beginning are those of a sudden, usually lightning-like, interruption of conduction in a certain segment of the cord. It is only rarely that the paralysis occupies an appreciable time (a few minutes to half an hour) in attaining its full extent; during this time violent pain is usually felt. In the most aggravated cases of extensive tubular hemorrhage the bleeding may last a number of days, and during the same length of time paralysis will gradually extend, usually from below upwards.

The extent of the symptoms depends of course upon the seat of the hemorrhage. When this is in the cervical and upper dorsal cord the arms, legs, and trunk are paralyzed, the arms frequently, however, only partially. In the arms alone, atrophy of the muscles accompanied by the reactions of degeneration will soon make its appearance, while the nutrition of the muscles will be but little affected in the trunk and the legs. Sensation is abolished up to the level of the area supplied by nerves arising from the affected segment of the spinal cord. The tendon and skin reflexes are abolished during the first few days, but later the tendon reflexes generally become exaggerated, as the total interruption of conduction in the affected segment is only rarely permanent. As regards the bladder, retention of urine is at first present, later there is difficulty in micturition or even ischuria paradoxa. The sphincter ani is paralyzed, but there is usually pronounced costiveness, so that soiling with fæces rarely occurs. If the seat of the lesion is in the upper portion of the dorsal cord, a unilateral or bilateral contraction of the pupil and narrowing of the palpebral fissure may also be noted. Occasionally priapism is present. If the hemorrhage has its seat in the dorsal region of the cord, the symptoms are the same, paralysis and atrophy of the muscles of the arm only being absent. When the seat of the lesion is in the lumbar region, the lower extremities alone are paralyzed and anæsthetic. The muscles of the lower extremities will then soon present atrophy and the reactions of degeneration. The tendon reflexes are usually permanently abolished in these cases. There

will be permanent incontinence of urine, the sphincter vesicæ being able to retain only a small quantity of urine. As a general rule impotence is present. The most varied grouping of paralysis and anæsthesia may also be present in apoplexy in the lumbar cord, according to the extent of the hemorrhage. For a more detailed account of the symptoms in cases of hemorrhage into the different segments the reader is referred to the section on injuries of the spinal cord. In hemorrhage into the conus medullaris we find paralysis of the bladder and rectum, impotence, and anæsthesia of the perineal region, the anus, and the genital organs, occasionally also of the posterior surface of the thigh.

The occurrence of trophic disturbances, especially bedsores, in the area of anæsthesia, and of disturbances of the function of the bladder, perhaps followed by cystitis and nephritis, is common to all severe cases. As a rule the temperature of the skin is somewhat raised in the paralyzed limbs; perspiration is abolished in the part.

In the worst cases death may occur from a spreading of the hemorrhage so as to involve the nuclei of the phrenic nerve, or from grave complications. In very rare cases it will occur a few minutes after the advent of the hemorrhage, which in such case is, of course, very extensive. As a rule the symptoms of a total transverse lesion, which are partly due to pressure by the clot or œdema in its vicinity, very rapidly disappear again. The functions of the bladder and rectum again return, the bedsores heal, and the paralysis is limited mainly to the most gravely affected parts, in which it is then, however, permanently established. This period of convalescence may continue a variable length of time, according to the size of the clot. Those muscles which remain permanently paralyzed, as a rule rapidly become atrophied and soon present complete reaction of degeneration, and after a time electrical irritability is altogether lost in them.

Owing to the preference of the hemorrhage for the gray matter, when it is situated in the cervical region of the cord, a more or less extensive atrophic paralysis of the arms remains; if it is situated in the lumbar region of the cord one of the lower extremities will remain paralyzed. If the posterior horns, as is usually the case, are also involved, there remains a permanent disturbance of sensation—sometimes only a partial one for pain and temperature.

In unilateral hemorrhage, the picture of Brown-Séquard's paralysis may be present, as has been observed by Minor; this consists in atrophy and paralysis of one arm and spastic paralysis of the lower extremity of the same side, and on the opposite side a loss of the temperature and pain sense; in that case the symptoms remind one very much of syringomyelia. If the destruction is con-

fined to one anterior horn, there may also result a pure atrophic paralysis of the extremity in question, without any disturbance of sensation whatever; if it is confined to the posterior horn, but involves a great extent of it, a hemianæsthesia for the sensations of temperature and pain alone may result.

When the hemorrhage is small and its action on the neighboring parts is slight, the paralysis may be partial from the beginning, and in that case the subsequent improvement will not be so marked as after total paralysis. There may also be present from the beginning a very extensive but incomplete paralysis—a paresis. Only in the most severe cases of traumatism is the total interruption of conduction in the cord a permanent one.

Diagnosis.

The diagnosis is easily made in those cases in which the etiological factors are characteristic and the beginning and further course of the disease are typical. Should a sudden grave paralysis, especially a paraplegia, occur after the reception of an injury, especially of the spinal column, in a previously healthy individual, and should it again disappear in the manner above described, so that finally the symptoms of only a circumscribed lesion remain, the diagnosis may then be made with certainty. It becomes difficult at once, however, when all these facts are not in accord, or when the previous history is defective, and we are in the presence of the final stage of the process. It is also more difficult if the symptoms point from the outset to a partial lesion of the cord.

The differentiation of a hemorrhage into the tissues of the spinal cord from one into the membranes, the so-called hæmatorrhachis, is very difficult, and in the beginning quite impossible. This can be readily understood, since a combination of both forms of hemorrhage will very probably occur after a severe trauma. In contradistinction to hæmatomyelia, the marked excentric pains and those of the back, the hyperæsthesias, and the stiffness of the spine are characteristic of hæmatorrhachis; on the other hand, in this affection disturbances of the bladder are usually absent, the paralyses and especially the hyperæsthesias are less marked, and the atrophy is by no means complete. Finally, perfect recovery usually results in hæmatorrhachis, but not in hæmatomyelia.

The differentiation from acute myelitis may be difficult. This, it is true, is of very infrequent occurrence, and paralysis probably never makes its appearance as rapidly as in hæmatomyelia. As a rule also prodromes are present, such as pain, spasms, etc.; the oc-

currence of fever also points to a myelitis. True myelitis usually occurs after infectious diseases or after poisoning; hæmatomyelia follows a traumatism. Improvement after a hemorrhage is more rapid and extends further than in myelitis.

Extensive softening of the cord due to the presence of a thrombus may, under certain circumstances, occur as rapidly as in the case of actual hemorrhage. As a rule, however, slight premonitory signs are present in this case, and there is no history of traumatism as an etiological factor; the thrombosis is commonly due to a syphilitic disease of the blood-vessels.

Poliomyelitis in the adult is usually initiated by a violent febrile movement, and its symptoms soon point to a number of foci of disease; as a rule some infectious disease has produced it. Cases of hæmatomyelia the previous history of which is unknown and which have run their course may be mistaken for the remains of an infantile spinal paralysis, when atrophic paralysis alone, without any sensory disturbance, is present.

As we have already seen, the picture of a hæmatomyelia which has run its course may very closely simulate a syringomyelia. The distinction between the two is rendered even more difficult by the fact that traumatism plays an important rôle in the etiology of syringomyelia, and that a true syringomyelia, according to Minor, may result from a hemorrhage in the spinal cord. The progressive course of syringomyelia is of diagnostic value.

If the symptom complex consists only in a partial hemianæsthesia, it would be necessary to differentiate a hæmatomyelia from hysteria. Here a history of paraplegia accompanied by bladder disturbance, and possibly decubitus in the early course of the disease, would decide in favor of hæmatomyelia.

Prognosis.

The prognosis of hæmatomyelia was considered bad in former times when cases of foudroyant myelitis and of progressive or relapsing thrombotic softening were not sharply differentiated from it. It was thought then that death was the usual result of a hemorrhage in the spinal cord. At the present time, the prognosis *quoad vitam* is quite favorable; when death occurs it is either during the very first stage, with terrible suddenness, or else during the first few weeks from grave complications. A fatal termination is naturally most frequent in cases of severe hemorrhages involving the entire thickness of the spinal cord, and also when the seat of the hemorrhage is in the cervical or lumbar portion of the cord.

After the first few weeks have been safely passed death rarely occurs, but neither does perfect recovery; the cure is always only a partial one and more or less marked permanent symptoms remain. In estimating the degree of improvement which will probably take place, we have to rely in great part upon the results of electrical examination; those muscles which show a complete reaction of degeneration will never recover, those showing simply a quantitative lowering of irritability are probably only indirectly injured and may recover. The patient's capability of earning a livelihood is of course dependent on the extent and the seat of the permanent lesion. If Minor is right, that a hæmatomyelia may pass into a syringomyelia, the prognosis will of course be more serious.

Treatment.

The first therapeutic indication after the occurrence of a hemorrhage of the spinal cord is rest, which should be as complete as possible and long continued. The patient is to be placed carefully in bed. The position on the abdomen is, if possible, to be taken, in order to avoid the occurrence of bedsores, and for the reason that an ice-bag may then be placed over the spinal column. The abstraction of blood by leeches is also to be recommended. All movement is to be prohibited for a long time. Straining at stool, sneezing, and coughing are to be avoided, if possible. With this avoidance of injurious things our therapeutic resources are really exhausted. Nature itself will take care of the improvement, as far as may be possible. During convalescence some benefit may be derived from a course of baths at one of the thermal-saline springs, such as Wildbad, Teplitz, or Nauheim. The application of electricity to the partially atrophied muscles may also be useful.

INFLAMMATION OF THE SPINAL MENINGES.

Pachymeningitis Cervicalis Hypertrophica.

By pachymeningitis is meant, anatomically, inflammation of the dura mater. Pachymeningitis cervicalis hypertrophica is an especial form, which, as the name implies, is found only in the cervical portion of the spinal cord.

Pathological Anatomy.

The disease consists in an inflammatory proliferation of the dura, leading to a thickening, an hypertrophy, of the latter. The proliferation takes the form of a slow deposition of fibrous tissue in concen-

tric layers, which may increase the dura to tenfold its normal thickness. This new formation compresses the spinal cord and the nerve roots which enter and leave it, and may even finally involve the whole thickness of the cord in atrophy and sclerosis. The thickening is greatest upon the posterior surface, and here adhesions with the extradural posterior vertebral ligament usually become established.

The etiology of the disease is quite unknown. Here, too, exposure to cold, overexertion, traumata, and various other things are alleged to be causative, without it being possible to prove the connection. Perhaps in some cases syphilis may be an etiological factor; and in others, chronic alcoholism.

Symptoms.

The clinical phenomena are divided into three stages.

The first, also called the neuralgic stage, is due to the increasing compression of the posterior roots, and is therefore distinguished by disturbances of sensibility. Violent pains are excited in the neck, in the occiput, and in the arms, and in these parts there are also subjective paræsthesiæ and sensations of numbness. Sensations of constriction may also occur in the upper part of the thorax. There is already stiffness of the neck; turning the head causes pain, and is therefore avoided. The cervical vertebræ are abnormally sensitive to pressure and percussion. In the arms the pains are localized by preference in the course of the ulnar and median nerves. They often possess a neuralgic character, occur without external cause in isolated attacks, and are very violent. Trophic changes sometimes appear in the skin in the form of roughness, exfoliation, or herpetic eruptions.

In this first stage of the disease, corresponding with the preponderant development of the process upon the posterior surface of the cord, the motor paths are not at all or but little affected. The only evidences that they are implicated are slight diminution in muscular power and irritative phenomena, such as twitching of the arms, trembling of the hands, and slight muscular tension in the upper extremities. The duration of the first stage is from two to six months.

The second stage corresponds with the extension of the pathological process to the anterior roots, and is marked by the appearance of paralysis and atrophy. These also are most pronounced in the regions supplied by the ulnar and median nerves, while that supplied by the radial escapes generally for a long time or even altogether. There develops a degenerative atrophy of the muscles innervated by the former nerves, *i.e.*, of all muscles of the hand, as well as of the

flexors in the forearm. Since the extensors of the hand preponderate on account of the still normal radial nerve, a position of the hand is caused which is quite typical of pachymeningitis cervicalis hypertrophica. It is overextended at the wrist, the first phalanges are extended, the second and third are bent—a position which suggests the hand of the preacher—and therefore is, in fact, called the "preacher's hand." Electrically the atrophied muscles manifest the phenomena of the reaction of degeneration. The sensory as well as the motor fibres of the ulnar and median nerves are affected. Besides the paræsthesiæ which are already present and which increase in intensity in the second stage, anæsthesia also develops, so that the hand is almost deprived of sensation, and the patients are no longer able to seize small objects with the fingers, and become very helpless.

In addition to the local affection of the ulnar and median nerves, the movement of the whole arm becomes impeded; it can be raised with difficulty at the shoulder-joint, and the movements of the elbow-joint are stiff and clumsy. Occasionally there may even develop a complete paralysis of one or both arms.

The third stage begins with the involvement of the nerve fibres of the lower extremity. This produces a motor weakness of the legs with more or less pronounced spastic phenomena, but without atrophy, because the cells of the anterior cornua of the lumbar cord—injury of which is the sole cause of atrophy of the leg muscles—are of course unaffected. But the compression of the cervical cord may lead to anæsthesia of the lower extremities, to vesical affections, and to decubitus.

The intense pains which characterize the first stage generally disappear almost completely in the second and third stages, the disturbances of motility becoming the most prominent features of the disease.

The duration of the disease is always several years. Cures have been reported, but in such cases the correctness of the diagnosis must be regarded with some doubt, for in general the disease must be considered incurable. Death occurs from intercurrent disease or from the decubitus or the affections of the bladder, with their dangerous sequelæ.

Diagnosis.

The diagnosis of pachymeningitis cervicalis hypertrophica is not easy. It is frequently confounded with other diseases, and generally the diagnosis long remains doubtful, certainty being reached only by observing the course of the disease.

Since the first stage is characterized only by pains, it can be easily

understood why it is generally diagnosticated as simple neuralgia of the occipital or of the ulnar and median nerves. The distinction from an idiopathic neuralgia is made possible only by the subsequent appearance of atrophy and of disturbances of motility which reveal the nature of the disease. The well-marked second stage may be confounded with several diseases.

Spondylitis cervicalis, as well as all tumors of the cervical portion of the spinal cord, especially syphilitic meningitis, may also be characterized by irritative phenomena in the arms, with consecutive muscular atrophy and paralyses. Spondylitis can be demonstrated by the sensitiveness to pressure of one of the cervical vertebræ, which is always present at a very early period, as well as by the appearance of tuberculosis in other organs. Ultimately also a deformity will manifest itself in the cervical spine, which is never the case in pachymeningitis cervicalis hypertrophica. But the distinction from other meningeal affections, as well as from tumors of the cervical spine, is very difficult, and often, indeed, quite impossible.

Progressive spinal muscular atrophy and amyotrophic lateral sclerosis can always be excluded with certainty, on account of the complete absence of disturbances of sensibility which is characteristic of both diseases, although the localization of the motor affections is very suggestive of pachymeningitis cervicalis hypertrophica. If there still remain doubts, the extension of spinal progressive muscular atrophy as well as of amyotrophic lateral sclerosis to the medulla oblongata with the production of bulbar symptoms will be decisive, since the latter, of course, never appear in pachymeningitis cervicalis hypertrophica.

It is often very difficult to exclude syringomyelia. It also begins generally in the cervical cord, and causes sensory irritative phenomena with atrophy of the hand muscles. Decisive for the diagnosis of syringomyelia are the typical partial paralyses of sensation. Moreover, the trophic affections which are the rule in syringomyelia, are exceptional in pachymeningitis cervicalis hypertrophica, while, on the other hand, in the latter the pains are, at first at least, by far the most conspicuous feature. Finally, the whole course of the disease in pachymeningitis cervicalis hypertrophica is much more rapid than in syringomyelia.

Multiple neuritis may also produce pains with atrophies, but the pains remain limited to the ulnar and median nerves; the stiffness of the neck and involvement of the occipitalis major and other nerves of the neck are wanting. In neuritis, too, the nerve trunks are sensitive to pressure or present thickenings; and finally, in multiple neuritis,

the cause, chiefly infection or intoxication (lead, alcohol, arsenic, etc.), can generally be made out.

If in the first stage the sensation of constriction of the thorax already described is very prominent, and one is forced to think of the possibility of an incipient tabes dorsalis. This can be decided only by the absence of the pupillary and patellar reflexes.

Treatment.

There is no effectual treatment for pachymeningitis cervicalis hypertrophica. A course of inunctions may be prescribed as an experiment. If these are not beneficial, we must confine ourselves to palliative measures.

Acute Leptomeningitis.

Acute inflammation of the soft meninges of the spinal cord generally involves at the same time the arachnoid and the pia, in rare cases the inner surface of the dura is also attacked, in equally rare instances the pia is hardly involved in the inflammation.

We shall not here consider tuberculous spinal leptomeningitis, for it is probably never met with in the absence of a similar disease of the cerebral meninges, and the symptoms of the latter will then predominate. Epidemic cerebrospinal meningitis will likewise be described elsewhere. We shall consider here only inflammation of the spinal arachnopia, which is usually purulent though sometimes serous in character.

Etiology.

The causes of acute purulent spinal meningitis are numerous. They are probably most clearly observed in those cases in which purulent inflammation of the meninges accompanies inflammatory or suppurative processes in the neighborhood of the spinal column or at more distant points. To these cases belongs the purulent meningitis following a traumatism of the spinal column and of the spinal cord. Here the infection may take place through an injury of the outer skin as well as through the thoracic and abdominal cavity, if an injury of the pleura or of the peritoneum and intestines has perhaps taken place. A purulent meningitis may of course also follow an operative procedure, and it was formerly frequently the result of operations in spina bifida. Furthermore, a bedsore may perforate the spinal column and be the direct cause of an inflammation of the meninges; it is also said that pleural and mediastinal suppurations have

extended to the arachnopia of the spinal cord in individual cases. As a rule an extensive purulent meningitis of the brain, especially one at the base of the brain, whether it be of a traumatic or otitic source, gives rise by extension to a spinal leptomeningitis. Of disease processes lying at some distance and acting by metastasis, purulent and septic inflammation is especially active. Thus a purulent cystitis may occasion a meningitis, this occurrence being most frequently observed in gonorrhœal cystitis or pyelitis. It is not necessary in these cases that a true gonorrhœal meningitis be present, but the inflammation of the meninges of the spinal cord may be produced by secondary carriers of infection, simple pus cocci. Not very infrequently the purulent meningitis in these cases is connected with an abscess of the spinal cord; indeed, there is but one case on record of abscess of the cord unaccompanied by purulent meningitis.

A peculiarly roundabout mode of origin is sometimes seen when purulent cystitis follows a severe traumatism of the spinal cord, and this cystitis in turn gives rise to a purulent meningitis by metastasis. More frequently even than a cystitis, cases of purulent bronchitis and abscess of the lungs have led to a metastatic purulent meningitis. Suppuration of other organs also may act in the same way. The acute infectious diseases are more rarely the cause of uncomplicated purulent meningitis, although the affection not very infrequently follows a pneumonia.

Of the surgical infectious diseases carbuncle of the back or of the nape of the neck leads with particular frequency to purulent meningitis; the latter has also been observed accompanying erysipelas. Finally it is not very infrequently a sequel to puerperal sepsis.

The so-called epidemic cerebrospinal meningitis occurs not infrequently sporadically, and may under certain circumstances confine itself to the spinal cord; it frequently accompanies pneumonia. According to Gowers an acute articular rheumatism may occasionally also lead to disease of the meninges of the spinal cord. Catching cold has been regarded as an etiological factor, and it is certain that spinal meningitis is sometimes observed as a direct sequence of a great cooling of the surface of the body, such as results from sleeping in the open air, or from a fall into the water.

Pathological Anatomy.

The extent of the inflammation in relation to the longitudinal axis of the spinal cord may be very variable. In cases of sporadic infectious inflammation, it is usually diffused over the whole cord. In cases of injury to the bones, or of abscess of the spinal cord, it may

be at first circumscribed, corresponding to the abscess or the injury, but later it always becomes diffused.

We may distinguish three stages of the disease, in an anatomical sense: (1) That of inflammatory hyperæmia; (2) that of the purulent inflammation proper; (3) that of disappearance.

The first stage is probably more or less artificial, as we rarely see it post mortem. The arachnoid, the pia, and even the inner surface of the dura are hyperæmic and not infrequently ecchymotic, the vessels are tensely filled, the cerebrospinal fluid is cloudy but not purulent, and as a rule hyperæmia of the cord will also be found.

The second stage is that which is most frequently met with at the autopsy. In this, the interstices of the arachnoid are usually filled with a thick, yellow, sometimes grayish pus; in rare cases there is only a cloudy fluid present, but in all cases the exudate is found microscopically to contain pus corpuscles. In severe cases the pia and the inner layers of the dura are also found to be filled with pus corpuscles; in light cases both may remain free of these. The blood-vessels are tensely filled, and their walls are studded with red and white blood corpuscles. It is remarkable how often the roots of the spinal nerves, which frequently lie in the midst of a collection of pus, and which, if we may judge from the symptoms, are certainly affected, appear to be anatomically intact. This is in accord with what we have learned in a previous section concerning the great resisting power of the nerve roots. In severe cases, however, the roots are affected; they become swollen and their nerve substance is disintegrated. The cord is involved in all cases of inflammation of the pia. We may find small irregularly distributed inflammatory foci, small abscesses, and also a form of marginal myelitis which establishes itself in the area of distribution of the marginal vessels which enter the spinal cord in its whole periphery; this latter may, under certain circumstances, give the impression of a combined system disease. If the pia remains intact, the cord may also remain perfectly healthy. The cerebrospinal fluid is increased and becomes turbid, containing flocculi of pus, but it is rarely macroscopically distinctly purulent. It, as well as the pus itself, contains micro-organisms which may vary according to the etiological factors; usually they are the common pus cocci; but in cases of epidemic cerebrospinal meningitis the pneumococcus has also been found.

In most cases death takes place in this second stage. If the duration of the disease is longer, or if recovery ensues, the inflammatory deposits may gradually become absorbed. A diffuse cloudiness and thickening of the meninges then usually remains; frequently adhesions are also formed among these, extending a longer or shorter

distance, and we may for a long time afterwards find remains of the inflammatory deposits. In other cases a local collection of fluid may take place at the level of the adhesions; this is called hydrorrhachis. It is possible also, though probably this is rare, that the calcareous plaques so frequently present in the arachnopia of the spinal cord have some connection with former inflammation. If the spinal cord has been markedly involved, permanent traces will undoubtedly be left behind in the shape of circumscribed or diffuse cicatrices, accompanied later by ascending and descending degeneration. Slight affections of the nerve roots will probably gradually disappear.

Symptoms.

We shall discuss here only those symptoms which are caused by a disease of the spinal meninges. These symptoms will be met with in the clinic only in those undoubtedly very rare cases in which the inflammation does not attack the cerebral meninges at all. When these are also involved, the cerebral symptoms usually predominate. We meet as a general rule with headache, delirium, or somnolence, and with affections of the cerebral nerves; when the cerebrum is involved, with convulsions and spasms; when the cerebellum is involved, with vomiting and disturbance of the heart and respiratory functions. The disease may occur at any age and in either sex. From the importance of the traumatic causes the male sex preponderates; the epidemic form quite frequently attacks children.

In the discussion of the symptoms of spinal leptomeningitis proper, we had better proceed from a case of this disease in its height, namely, in the second stage, that of formation of pus in the meninges. The symptoms present at this time, leaving aside the fever which is probably always present and the marked constitutional disturbance, may be divided into the symptoms caused by the disease of the meninges themselves, by that of the spinal roots, and by that of the cord. The first consist in the most violent backache, which becomes unbearable with every movement, even the slightest. The seat of this pain is, according to the extent of the disease, in the whole back or limited to certain points. As a rule the spinous processes are also tender to firm pressure over the site of the inflammation.

The symptoms depending on an affection of the roots may be divided into symptoms of irritation and those of paralysis. The first are much more constant, and for this reason of more practical importance than the latter, which in a great many cases are not present at all, and in others may come on very late. A lesion of the posterior roots is manifested by tearing pains radiating into the limbs, by

girdle pains, or by marked hyperæsthesia of the skin, limited to the area of distribution of the injured nerve root. As a rule the muscles are also quite painful to pressure, and active as well as passive movements increase these pains. The motor symptoms of irritation consist mostly in more or less extensive muscular contractions. These are the cause of the very characteristic rigidity of the neck, which occasionally is so marked that the occiput lies in close contact with the nape. Total opisthotonos may also occur in extensive meningitis. It is not altogether impossible that these forms of contraction are in part voluntary, though unknowingly so, being due to an effort to fix the spinal column as much as possible and to avoid its every movement. Other contractions of this kind are undoubtedly not dependent on the will, and are either purely reflex, that is, they are dependent on an irritation of the sensory roots, or are perhaps due directly to an irritation of the anterior roots. To these belong the contraction of the abdominal muscles, which produce the well-known boat-shaped retraction of the abdomen, and also contractions of the arms and legs. The very painful recurring spasms of the upper extremities, which are seen especially in cases of circumscribed meningitis, are undoubtedly due to a direct irritation of the anterior roots. A rhythmical and rapid tremor is also seen, especially in the upper extremities. The so-called Kernig's symptom should also be classed among the muscular contractions; it consists in an inability of the patient to extend the knees, the flexors of the legs being too short. Paralytic symptoms due to the lesion of the nerve roots, namely, atrophy of the muscles and circumscribed anæsthesia, are of rare occurrence. This is due to the relatively strong resisting power of the nerve roots, also to the fact that, although the inflammation may be sufficiently intense to cause these root symptoms, it is nevertheless not so pronounced as to destroy all the fibres of these roots. Marked anæsthesia and paralysis should therefore always make us suspicious of an extensive involvement of the cord. More frequent on the other hand are trophic disturbances, especially of the skin. Thus herpes zoster is sometimes seen; in other cases large bullæ occur from slight causes, as, for example, when one lower extremity has been pressing, even for a short time only, against the other; these bullæ look as if caused by a burn (taches spinales). Symptoms of vasomotor disturbances may also be present; thus a slight touch or the prick of a needle may cause the skin to puff up in high wheals with a red zone.

The participation of the cord itself may be shown in various ways, such as disturbances of the bladder or rectum or an exaggeration of the tendon reflexes, going on even to distinct clonus of the patellar and Achilles tendons; the skin reflexes also are usually increased; and

finally there will be extensive anæsthesia and paralysis, as in myelitis and injuries of the spinal cord. This paralysis is usually at first spastic in character, but in very severe cases it may be of the flaccid form and be accompanied by loss of the tendon reflex. In these cases we are usually unable to say with certainty which of the symptoms are due to the root lesion and which to the spinal-cord lesion. At this time decubitus may also make its appearance. Of course, the distribution of all these symptoms varies according to the seat and extent of the meningitis. As the latter is usually diffused so are also the symptoms; pain and contractures make their appearance now here, now there, and the entire spinal column is more or less rigid. In cases of circumscribed inflammation, when the seat of the lesion is in the cervical or upper dorsal region, the pain will be most marked in the arms, contraction of the pupils and narrowing of the palpebral fissures may appear, and as a rule obstruction to the respiration is quite marked. When the seat is in the dorsal region, the symptoms are confined chiefly to the trunk, and when it is in the lumbar region the legs are principally affected.

Course.

The course of the disease is usually acute, sometimes it is hyperacute. It is probable nearly always initiated by a chill, which is in most cases followed by a continued high fever of a remittent character. All the symptoms rapidly attain a high degree, a marked stiffness of the muscles of the neck in particular is frequently present by the end of twenty-four hours. Death may take place within a few days, but as a rule, the encephalon is involved in these cases and the fatal termination results from paralysis of the heart or from asphyxia. In other cases the disease drags on for a few months, and the grave symptoms of cord involvement make their appearance gradually. In the epidemic form the disease may take a different course. Here the fever may cease after a few weeks, the pain and contractures disappear, the appetite returns, and the patient improves; thus, after weeks of apparent convalescence an acute relapse may occur; this may again be followed by improvement, and the patient may finally die from exhaustion after these alternations of relapse and improvement have continued for many months. Recovery is nevertheless possible and not infrequent in this form of the disease. Frequently of course the termination is in partial recovery only. If grave lesions of the nerve roots or of the cord have occurred, circumscribed paralysis and anæsthesia, or perhaps a spastic paresis with slight disturbances of the function of the bladder, are certain to remain. A permanent rigidity of the muscles of the back may also persist.

Diagnosis.

The diagnosis is not difficult, if the characteristic symptoms of the disease are distinctly marked. These are fever, pain in the back, and opisthotonos, pain and hyperæsthesia of the skin, muscular pains and spasms, and possibly a spastic paresis and disturbance of the bladder function. The diagnosis is rendered still more certain if symptoms of irritation precede those of paralysis, if distinct symptoms of disease of the cerebral meninges are associated with those of spinal leptomeningitis, and especially if certain recognized etiological factors, such as traumatism, purulent processes in other organs, or infectious diseases, are present.

The differential diagnosis from hæmatorrhachis may be more difficult. The symptoms are the same in both diseases, but those of hæmatorrhachis must follow immediately after the traumatism, while there would always be an interval of a few days before the appearance of an infectious meningitis. The fever will be absent in simple hemorrhage.

The distinction of a meningitis from a myelitis will, of course, be difficult in those cases in which the spinal cord is materially involved in the inflammation, and it may even be impossible under such circumstances. Very severe pain, especially pain and stiffness of the back, will always indicate an involvement of the meninges, and we may take for granted that we have primarily to deal with a meningitis when all these meningeal symptoms at first existed alone, and paralysis, indicating a lesion of the spinal cord, followed later. Formerly the differentiation of spinal meningitis from tetanus was often difficult. The possibility of a diagnosis lies here in a knowledge of the etiological factors. In both diseases opisthotonos and tonic spasms are noticeable; in tetanus, however, the fever is nearly always absent, especially the initial fever, radiating paræsthesias and hyperæsthesia of the skin are not present, and the pains of tetanus are produced only by the continued muscular tension: tetanus nearly always begins with trismus, which may be present also in cerebral meningitis, but usually appears at a later period; in tetanus symptoms due to the cerebrum, especially delirium, coma, and convulsions, are absent; on the other hand, a marked difficulty of deglutition and dyspnœa make their appearance in tetanus earlier and are more severe. The reflex irritability is increased in tetanus to a degree that is probably never seen in meningitis; even moving towards the patient's bed or slight stroking of the hand over the skin is sufficient to bring on the most violent tonic spasms.

Certain hysterical manifestations may for a time arouse a sus-

picion of the presence of meningitis; we may find headache, vomiting, rigidity of the muscles of the neck, or even convulsions, or a slow pulse and seeming stupor. In such cases only a very careful examination will preserve us from error, by showing us that the stupor is only apparent, that the symptoms may be influenced psychically, that sleep is undisturbed, etc.

Of especial importance is the distinction of a purulent meningitis from a tuberculous one. Purulent meningitis usually comes on in a turbulent manner, the first day being marked by a stiffness of the neck, to a degree which is hardly ever found in tuberculous meningitis; the fever is also higher. In one of the writer's cases the diagnosis was determined by puncture of the spinal meninges, which yielded a turbid fluid containing small pus corpuscles and a great number of cocci. In tuberculous meningitis the fluid from the puncture is nearly always clear and may contain tubercle bacilli.

Prognosis.

The prognosis of acute purulent leptomeningitis is always grave. As a rule death is the inevitable result after a longer or shorter period. In individual cases the prognosis depends less on the extent of the meningitis (since this disease always shows a tendency to become diffuse) than on the etiological factors. If spinal meningitis is due to an injury, to a suppuration in the neighborhood of the spinal column, or to a metastasis from an abscess lying at some distance, the result cannot be other than fatal. Meningitis occurring as a sequel to an acute infectious disease (typhoid fever or rheumatism) is usually of very bad prognosis. The prognosis is materially better in those cases of autochthonous purulent spinal meningitis, if we may call it so, which occur sporadically, but resemble in their symptoms epidemic cerebrospinal meningitis. Here we meet with a whole series of cases ending in perfect recovery, or another in which only a partial cure results. The prognosis becomes more favorable after the first few days of the affection have passed, and it is more favorable still after the fever and pain have subsided and convalescence has begun. Even then, however, we must be very guarded in our prognosis, remembering the frequent relapses which may cause death directly or indirectly by asthenia or other complications. Thus in a case occurring in a child in the writer's practice, a purulent inflammation of the meninges of the brain and spinal cord had been cured, but a tuberculosis of the lungs soon appeared and ran a rapid course, and to it the little patient succumbed. The prognosis is also rendered uncertain by the possibility that permanent paralysis, muscular

atrophy, or other sequel may remain. This is indeed always to be expected, if the spinal cord has been involved to any great extent.

Treatment.

We can do little more in these cases than to sustain the strength of the patient by careful nursing, and to try as much as possible to assist him in his battle with the disease. Rest in bed in a cool, airy, darkened room is always necessary. The bed should be smooth, and the patient should, if possible, be laid on a water cushion. It is theoretically to be recommended that he lie on the side or better still on the abdomen, but practically it will be possible to retain the patient in this position for a short time only. The food should be strengthening, but not stimulating; cooling drinks, such as lemonade or soda water, are indicated, but alcohol is permissible only when there is danger of collapse. In severe cerebrospinal meningitis, especially in children, feeding must sometimes be carried on by the stomach tube. Of importance is also the minute care of the skin, above all scrupulous cleanliness.

Antiphlogistic and derivative remedies are said to be of direct therapeutic value. It is well not to expect too much from these, and not to make use of them too energetically. We may locally employ venesection or wet-cupping, or make ice applications to the spinal column. Inunction with blue ointment, 1.0–4.0 (gr. xv.–℥ i.) a day, has been recommended, as also the internal exhibition of calomel. Hydrotherapeutic measures would seem to promise good results; they are easy of application, especially the moist pack, but hot baths are hardly to be recommended on account of the difficulty experienced in moving the patient. For the relief of violent pain morphine should be freely used with proper precautions. Of hypnotics chloral hydrate still retains its place.

Chronic Leptomeningitis.

If we except pachymeningitis cervicalis hypertrophica, pachymeningitis interna hæmorrhagica, and syphilitic spinal meningitis, primary chronic inflammation of the meninges of the spinal cord is undoubtedly a very rare disease, even admitting the fact of its existence. In the first two of the above-mentioned varieties of spinal meningitis the names themselves denote that the inflammation is one principally of the dura; and in syphilitic meningitis both the dura and pia are usually affected at the same time. The existence, however, of a primary, simple chronic inflammation of the arachnopia is

very doubtful. We frequently meet with such as a secondary disease in tabes dorsalis and progressive paralysis, in myelitis, and in tumors. It is, however, of no more clinical importance here, although probably it does not occur without symptoms, than it is in senility and in chronic alcoholism, in which the exudate on the membranes is looked upon as due to a chronic inflammation, of which there is nevertheless no clinical evidence. It was formerly believed that a chronic meningitis not infrequently followed an acute one, but in fact the result of acute spinal meningitis is either a perfect recovery or an acute relapse after a longer or shorter period of improvement.

At all events, chronic leptomeningitis was formerly greatly overrated as to its frequency, and the diagnosis was often incorrect. Thus Gowers mentions that the symptom complex, which is now regarded as that of spastic spinal paralysis, was formerly nearly always looked upon as a chronic meningitis. The same thing has undoubtedly taken place in hysteria, and also in neurasthenia, and especially in those cases which were formerly called spinal irritation, and which are manifested by the presence of acute pain on pressure over the vertebral spines and violent backache. Both the latter are also main symptoms of neuroses occurring after railroad accidents and other traumatisms, which at all events are related to hysteria and neurasthenia, and which formerly were nearly always supposed to be due to a chronic meningitis. Many cases of chronic neuritis were also probably looked upon formerly as cases of meningitis, and the same is true of many cases of myelitis, in which the meninges may be involved, and which may then present quite marked symptoms of irritation in the sensory region, especially pain and rigidity of the spine.

The existence of this disease being then so very doubtful, we must regard the *symptoms* that are given in the text-books as purely imaginary. If the disease were present it would, no doubt, be manifested by backache and rigidity of the spine, radiating pains, less intense than in acute inflammation, spastic paresis of the lower extremities, and possibly circumscribed muscular paralysis and atrophy. The spinal column in its entire extent, or at individual points, would be painful to pressure. Anatomically the findings would correspond with those of the convalescent stage of acute meningitis, that is to say, cloudiness, swelling, and thickening of the pia, swelling and finally shrinkage of the nerve roots, marginal sclerosis, etc. We should beware above all of mistaking it for any of the functional diseases above mentioned. It is important to remember that the pain in hysterical rachialgia is usually more marked when the skin over the spinal column is lightly touched than when actual pressure is made on the spinous processes.

For the treatment of this hypothetical affection a course at one of the many thermal springs has been recommended, followed perhaps by a mild cold-water cure; internally, we may give iodide of potassium. In the event of recovery under this treatment the probability will be that we had to do with a case of neurasthenia or hysteria, and not one of chronic spinal leptomeningitis.

POLIOMYELITIS ANTERIOR.

Poliomyelitis Anterior Acuta Infantum.

Synonyms.—Infantile spinal paralysis, myelitis of the anterior horns, acute anterior tephromyelitis, essential or atrophic paralysis of childhood.

Definition.

The affection begins suddenly, accompanied by violent febrile movement, also frequently with marked cerebral symptoms, especially convulsions; at other times it may occur without any constitutional symptoms whatever. There is a rapid appearance of paralysis, usually extensive in the beginning, sometimes involving the whole muscular tissue of the trunk and extremities, and even more frequently under a paraplegic form involving both legs, with loss of reflexes. There is, however, no disturbance of sensation. The sphincters are not often implicated and trophic disturbances of the skin, especially decubitus, are not encountered. The general symptoms are of short duration. The paralysis always shows its greatest extent at the beginning, and is never progressive after the first few days. Recovery takes place in such a manner that power is gradually restored in one muscle after the other and even in entire extremities, and finally the paralysis is confined to one limb, to large groups of muscles, or to single muscles; a perfect recovery, however, is met with only in rare cases. The muscles which remain permanently paralyzed become greatly atrophied, the muscles and their nerves at first showing a complete reaction of degeneration, and later an absence of all electrical irritability. The paralyzed portions of the limbs also show slight vasomotor disturbances of the skin. The growth of the bones also does not proceed in a normal manner. As a result of the muscular paralysis, of impediment in the use of the limbs, and of the retardation of osseous development, deformities will arise later, especially in the lower extremities and the spinal column (clubfoot, scoliosis, kyphosis, etc.). In other respects the

general health, as well as the bodily and mental development, remains permanently good. The disease is confined chiefly to early childhood, between the ages of one and four years; after this it is comparatively rare.

Anatomically we have to deal with an acute inflammatory process, which confines itself in a great measure, though not altogether sharply, to the gray matter of the anterior horns, leading to the destruction of their ganglionic cells and medullary plexuses with resultant changes in the spinal roots, nerves, and muscles. The inflammation, from the standpoint of the pathologist, is an interstitial one; it arises from the blood-vessels and involves pretty acutely the area of the artery of the anterior medullary groove. We are therefore dealing with a *myelitis* of the gray matter of the anterior horns, localized mainly in the lumbar and cervical enlargements.

History.

As the clinical picture of infantile spinal paralysis is such a sharply defined one, it seems almost certain that it must have early attracted the attention of good observers as an independent affection. We find in fact, in the literature of the eighteenth century, reports of individual cases which belong here. J. v. Heine of Cannstadt, is, however, regarded by general consent, and with perfect justice, as the first to have established the existence of infantile poliomyelitis anterior. In a work published in 1840 (a second edition of the same appearing in 1860), v. Heine made a report of this affection with especial reference to the deformities which accompany it. This monograph is clinically, even to this day, an exhaustive treatise. Further clinical advance then followed very rapidly. Notable contributions were made in 1851 by Rilliet of Geneva, who gave to the disease the name of essential paralysis of childhood. Duchenne of Boulogne wrote most exhaustively on the functional disturbances and deformities induced by paralysis of the individual muscles, and was the first to call attention to the absence of reaction to the faradic current in the paralyzed muscles. This fact was further developed by Erb, who proved by experiments with the galvanic current that we have to deal with a complete reaction of degeneration in infantile spinal paralysis, just as we have in injury to the peripheral nerves. To Seeligmüller belongs the special credit of having given a full explanation of the origin of the contractures and deformities in this disease.

Of a much later date than our clinical knowledge of infantile spinal paralysis is that of the anatomical basis of this disease. It is true

cerebral symptoms, convulsions and cranial-nerve palsies, do not enter into consideration in this connection.

Symptoms and Course.

Spinal paralysis of childhood may, according to its course, be divided into three stages: 1. The prodromal period, and the acute beginning of the disease, the initial stage; 2. The period of paralysis in its most diffused stage; 3. The period of the gradual amelioration of this paralysis as far as it will go, and the determination of the permanently paralyzed groups of muscles, including the sequelæ of the paralysis (contractions, deformities). These periods are of course not sharply marked, but usually merge into each other. Distinct prodromal pains are very rare; sometimes the children will complain of weakness of the limbs, become listless, and lose their appetite. As a rule, however, they are suddenly overtaken by the disease while in perfect health. It is possible to distinguish four forms of the initial stage, characterized as febrile, or febroneurotic, or as purely neurotic, or possibly there may be no particular symptoms; more logically expressed, the initial stage is absent. We may confess here that we know very little about the initial stage from direct medical observation, as medical assistance is usually invoked only after paralysis has already occurred; only the more severe nervous symptoms of the initial stage are likely to come under medical observation. As a general rule, however, we shall have to fall back on the previous history.

The febrile initial stage is most frequently met with. It is usually accompanied by gastric symptoms, often by vomiting. In a few closely observed cases it was found that the temperature had risen above 39° C. (102.2° F.). Fever usually continues only a short while—twenty-four hours—at most three to four days (Medin).

In the febroneurotic initial stage the fever is accompanied by marked cerebral symptoms, most frequently spasms are present, and these usually in the form of general convulsions; more rarely they involve only one side or a single extremity. One attack only may occur, or a status epilepticus may remain. In this case the children may sometimes die, and then the diagnosis is very difficult to make; the latter was, however, determined post mortem in a number of Medin's cases. A comatose or stuporous condition may take the place of the spasm. It is only in rare cases that fever is absent and neurotic symptoms alone are present.

Cases in which distinct initial symptoms are absent are not at all rare, at least according to the history furnished by the parents. The

child has been put to bed on the previous evening in perfect health, has slept peacefully during the night, and in the morning the paralysis is noticed. In very young children, or when no close observation has been made, the paralysis may occasionally not be discovered by the parents until a long time after its occurrence.

The paralysis itself—the second stage having now been reached—is generally a very extensive one in the beginning. In many cases, the trunk and all four extremities are involved, frequently also the muscles at the nape of the neck; and in cases in which the paralysis is confined to the lower extremities, there is at least a total paraplegia. As the beginning of the paralysis is hardly ever observed by the physician, we have no positive knowledge of the manner of its development; its appearance is at all events very sudden—in some cases nearly in an apoplectiform manner. It has only rarely been observed that one limb, for example, was paralyzed in the morning, and another in the evening. As a general rule it may be assumed that after distinct paralytic symptoms have existed in a case for nearly twenty-four hours, a further extension of the latter need not be feared, the disease has then reached its acme, and a change for the better only is to be looked for; the paralysis therefore is in no case a progressive one.

Cases in which the paralysis from the beginning involves only limited muscular areas, as one leg, the muscles of the sacral plexus, the shoulder and arm, are, as already mentioned, of rare occurrence. In these cases of partial paralysis, the general rule holds good that in the arm the proximal, in the leg the distal, muscular areas are usually paralyzed, although exceptions to this rule may of course be met with. It is important to remember that paralysis of certain cerebral nerves may also occur in the course of a "spinal" paralysis of childhood. Most frequently the hypoglossus or the facial, or the facial and abducens are affected. We have met with cases of paralysis of the facial nerve which were very similar, especially in their local symptoms, to a poliomyelitis acuta, but in which there was no spinal paralysis whatever. These cases are probably closely related to those of extensive encephalitis recently described by Oppenheim. We have seen a similar case in which there remained a slight facial paralysis. Medin also has recorded a few instances of facial paralysis among his cases; Redlich found in such a case foci of disease in the pons and medulla oblongata. These cases are also important from a theoretical point of view, because they demonstrate the presence of disease of the general nervous system in infantile spinal paralysis.

The paralysis in acute poliomyelitis is at the outset a flaccid one,

the reflexes in its whole area being absent. At first there is, of course, no atrophy, and the disturbances of reaction to the electric current are probably very slight during the first few days. Seeligmüller has, however, found marked lowering of electric irritability as early as the third day. Sensation is always preserved, as far as this can be determined in little children; pain is said to be present occasionally. The bladder may be affected in the initial stage, although, of course, this is a difficult matter to determine in very young children, and the same may be said of disturbances of function of the rectum. There is never any permanent involvement of these organs in the later stages of infantile paralysis. As a rule the general health is good. Decubitus is never produced. This severe stage of the paralysis, consisting in the worst cases of a total paralysis of most muscles whose innervation proceeds from the spinal cord, lasts only a short time, usually not longer than a couple of days. Then the first movements return generally in the toes or in the fingers.

At first improvement is rapid, very soon one whole extremity, or both upper ones for example, again recover their motility. Usually the muscles of the nape of the neck and also those of the trunk rapidly improve. By the end of the first six weeks only those muscles remain paralyzed which will be permanently disabled. Recovery is, however, not yet completed, but its progress from this time on is slower. Finally only a portion of the muscles of the extremity which was chiefly affected remains paralyzed, while another portion goes on to recovery. In this manner convalescence may continue into the third quarter of the year from the date of the beginning of the paralysis, but after this the affection usually remains stationary; in other words, those muscles which have not regained their functions by the expiration of nine months will never afterwards regain their power.

It may, as a general thing, be said that the more extensive the paralysis, the more rapidly in the beginning will be the improvement. If, on the other hand, the paralysis was a circumscribed one from the beginning, then convalescence also be slow. This is dependent on the fact that in the first case the anatomical changes, although diffuse, are severe only at one point, while in the latter case they are confined to a narrow field, but are pronounced in this area. For this reason those rare cases in which the paralysis does not improve at all, but persists in the stage of its greatest extension, are marked usually by a paralysis which is circumscribed from the beginning. It is questionable whether improvement in infantile spinal paralysis can ever progress to a perfect recovery; the possibility of this cannot be denied *a priori*, but at any rate it must be of very rare occurrence; it is difficult to conceive of the possibility of a cure in the ana-

tomical sense. We are at this day aware that the motor centres of most muscles occupy more than one spinal segment, so that a paralysis of any given muscle obtains only when its entire nerve centre is destroyed. Poliomyelitic foci, which confine themselves to one segment, therefore, need not cause a permanent paralysis. This latter condition, however, occurs unfortunately very rarely, as the branches of the central artery distribute themselves over numerous segments. An apparently perfect cure may perhaps sometimes occur, when a lesion is present in the dorsal region of the cord, involving at first the muscles of the cervical or lumbar enlargements, and later concentrates itself altogether in the dorsal region, thus giving no evidences of its presence. Practically, however, it may be said that in all cases of infantile spinal paralysis a definite paralysis will remain behind in some part of the body.

The grouping of these definite forms of paralysis is of course exceedingly diverse, one is inclined to say arbitrary, as it depends on the site and the size of the lesion, and of course also upon the number of lesions remaining. Seeligmüller has compiled a table of the individual varieties, according to the frequency of their occurrence. Paralysis of the lower extremities is much more frequent than that of the upper. Paralysis of one lower extremity only is most frequently met with, and then usually does not involve the entire limb, being often confined to the muscles of the leg, to the flexor or extensor muscles of the foot, or perhaps to the muscles of the thigh, those of the leg remaining normal. Total paralysis of all the muscles of one leg is very rare. We have seen the long flexor muscles of the toes remain normal, with relative frequency, in cases of nearly total paralysis. Some one or more of the muscles of the hip joint also usually escape. Paralysis of both limbs frequently occurs, but it is hardly ever entirely symmetrical; thus the muscles of both thigh and leg may be involved in one limb, while in the other only those of the leg are affected. A paralysis of all four extremities, paraplegia, or a crossed paralysis, as of the right leg and the left arm for example, is rare. Hemiplegic forms of paralysis occur very rarely; the rarest of all forms is a cervical paraplegia. We see not quite so infrequently a paralysis confined to one tibialis anticus muscle. Permanent forms of paralysis of the muscles of the trunk are more frequent, but those of the flexors of the neck and head or of the diaphragm are very rare.

Although there is apparently little order in the distribution of the paralysis, this is changed when we come to study the grouping of the permanent forms of paralysis. To Remak belongs the credit of having been the first to point out, from purely clinical observations, that certain combinations in the forms of the paralysis typically recur.

He also came to the conclusion that these types must have their origin in the anatomical grouping of the motor centres of the muscles in the individual diseased segments of the spinal cord. Further clinical observations, especially in cases of injury of the spinal cord, as also experimental researches, made especially by the English, have shown that in affections of the upper portion of the cervical enlargement we have paralysis of the deltoid, infraspinatus, biceps brachialis, and supinator longus muscles. Should the affection of the cervical portion of the spinal cord travel downwards, the extensors of the hands and fingers will be first affected; should it have its seat in the lower portion of the cervical enlargement, the flexors alone may possibly be paralyzed. Paralysis of the quadriceps is very frequently combined with that of the adductor muscles, although the sartorius, which arises much higher, escapes with remarkable frequency. On the other hand, the tibialis anticus is not rarely involved in paralysis of the quadriceps femoris. This muscle again sometimes remains normal, while all the muscles of the leg are affected, or finally it may be the only one paralyzed. The extensor communis digitorum is generally paralyzed together with the peronei muscles, the muscles of the calf remaining intact, and when the latter are involved it frequently happens that the peronei escape.

As before mentioned, all these types are fully explained by what we now know as to the grouping of the muscle centres in the individual segments of the spinal cord, the anatomical details of which it is unnecessary to enter upon here. This rule, however, is of course not an absolute one, for the extent of the individual lesion is naturally not always the same, and quite irregular offshoots may branch out from the main focus and alter the symptomatology very considerably; the grouping of the paralysis will also depend upon the size of the motor centre of each individual muscle, as one of slight extent is naturally more easily destroyed than a long one. The paralysis is of the relaxed variety from beginning, and so remains even in the muscles which are permanently paralyzed. Neurogenous contractures never occur, and those contractures which are frequently met with later on are, as we shall see, of an altogether different nature. All the reflexes which are dependent on the permanently paralyzed muscles remain definitely absent. As regards the sphincters and the general sensibility, nothing need be added to what has already been said.

The trophic disturbances, especially those relating to the muscles, are of very great importance. The muscles given over to permanent paralysis usually very rapidly become atrophied. This atrophy may generally be demonstrated within a few weeks after the attack, and it

then makes rapid progress. When the muscles of an entire extremity remain permanently paralyzed, they may, by the end of from six to nine months, be so far destroyed that there seems to be nothing but bone covered by the skin. Those muscles which recover during the first few weeks do not become atrophied, but those portions of the muscle whose restoration is deferred for a long time, many months perhaps, will become more or less atrophied according to the duration of the paralysis.

The changes in electrical reaction will go hand-in-hand with the trophic disturbances of the muscles. In the greatly affected muscular areas, exactly as in grave peripheral paralysis, there may be demonstrated, probably after twelve to fourteen days, sometimes a little later, a complete reaction of degeneration. Both intra- and extramuscular irritability to the faradic current will be absent, as well as the extramuscular irritability to the galvanic current. On the other hand, the direct galvanic excitability of the muscles is increased, and we frequently obtain the formula, AnCC>CaCC, but the contractions are slow. The muscles which rapidly recover do not show any change in their electrical irritability; those which remain paralyzed for a long time, but eventually recover, generally show a slight diminution of excitability to all forms of electricity, but without qualitative changes. It may thus be seen of what importance an electrical examination is in the diagnosis and especially in the prognosis of infantile spinal paralysis; for it may be said with absolute certainty that those muscles which show complete reaction of degeneration after the fourteenth day will remain permanently paralyzed, and that, on the other hand, all others, even those with a marked but simple diminution of excitability, will recover.

In the permanently paralyzed muscles the exaggeration of galvanic irritability disappears at about the second or third month, and then this irritability gradually decreases more and more; but it is very characteristic of infantile spinal paralysis that the remains of an intramuscular galvanic excitability may still be demonstrated for a long time in the paralyzed muscles.

The trophic disturbances of the bones have been mentioned above. We have to deal mostly with a retardation of growth in their length; the bones, however, also remain thinner, and their ridges for the muscular attachments are absent. This retardation of growth is found in the paralyzed extremities, but does not altogether conform to the gravity of the muscular paralysis. Seeligmüller has seen one case of excessive growth of bone in its length. True trophic disorders of the skin are not observed, but vasomotor disturbances are always found. Generally the skin of the paralyzed limbs is cyanotic and colder

than that of those not paralyzed; occasionally also it is slightly œdematous.

Finally, the contractures and deformities occurring as a sequel to the paralysis are of classical importance. The two conditions overlap each other. We designate as contractures the distortion of the position of the limbs in the early period, when they can readily be corrected; while we speak of deformities only when the abnormal position cannot be rectified without having recourse to heroic measures, for reasons that will be discussed below. Besides these true articular deformities we meet, especially in the worst cases, with the so-called flail joint. The deformities, like the paralysis, are most frequently found in the lower extremity, especially in the foot. Most generally we find talipes equinovarus, next to it equinus, frequently accompanied by pes cavus; more rarely we see pes planus, valgus, or varus, and much less frequently talipes calcaneus, which may be divided into the more frequently occurring talipes calcaneovalgus, with accompanying cavus and talipes calcaneovarus. At the knee-joint we most frequently meet with genu recurvatum, eversum, or inversum—very rarely with a contracture of the flexor muscles. At the hip joint we frequently find a flail joint (*jambe de polichinelle*); contractures in the position of abduction or adduction with flexion and rotation are also encountered. In these cases luxations of the head of the femur may also occur.

In the shoulder we frequently find that the head of the humerus has left the glenoid cavity, the so-called paralytic subluxation. In the hand, flexion of the fingers, or extension of both fingers and hand, may be observed. In the hand and fingers contractures only but no actual deformities are found.

There are three factors which are concerned in the production of the deformities of infantile paralysis; these are in the order of their importance as follows: In the first instance we have the primary voluntary contraction of the non-paralyzed muscles of the joint, which, not being corrected by the opposing paralyzed muscles, become permanently contractured. In this manner the deformities are produced, especially in cases in which only certain of the muscles which move a joint are paralyzed. Under the second heading may be placed the static conditions especially urged by Volkmann, namely, the simple weight of the paralyzed limbs, the pressure of the bed clothes, and the result, especially in the foot, of the faulty position caused by the active contraction, for example, talipes varus or valgus or calcaneus, which is continually increased by the act of walking. These static conditions must of necessity act alone if all the muscles of a joint are paralyzed. They act together along with the contraction

of the muscles in cases of paralysis of a portion only of the muscles of a joint. They may here, as previously mentioned, increase the effect of the muscular contractions, or sometimes may antagonize it, as, for example, in the case of a pes equinus in which the action of the muscles is not too powerful. It goes without saying that these static causes of deformities act especially in long neglected cases. It cannot, however, be emphasized too strongly that of the two mentioned factors the first—the muscular traction—is by far the more important. This may be seen particularly when it causes changes in the position of a limb directly opposed to the weight of the extremity; thus, for example, in pes calcaneus, in cases in which the children have never walked, in flexion of the knees, in flexion and abduction or adduction of the hip-joint, in extension of the hand and fingers. It is a consideration of these cases which has in particular led to the final rejection of the teaching of Volkmann, who acknowledged only static conditions in the production of deformities.

The third factor consists in permanent nutritive shortening of the contractured muscles, shortening and deformity of the tendons and ligaments, and changes in the surfaces of the joints. In this manner the retardation in the growth of the bone is also brought about. At this stage the distortions of position are not easy to correct, for the deformity is already an accomplished fact. As before stated, rigid deformity of a joint does not usually occur when all the muscles moving it are paralyzed, for then a flail joint is the result. It is not to be expected, when we consider the variety of the individual factors causing the deformity, that in each individual case, with a similar grouping of the paralyzed muscles the same anomaly of position should occur; in other words, we cannot always with certainty tell, from the deformity alone, which muscles are paralyzed, for our calculations are frequently overthrown by the other influences above mentioned. As, however, the traction of the non-paralyzed group of muscles is the chief factor in the production of the deformity, we are able from a study of direction of this deformity to arrive at some conclusion concerning the seat of the paralysis. But we must remember that nearly every individual deformity can be produced not only by one but by several forms of paralytic contracture, and we should therefore always control any conclusions based upon the data furnished by the deformity by other methods of examination, especially by an electrical examination. Individually, the following may perhaps be said of the various deformities. Pes equinus may be occasioned by simple static factors if all the muscles by which the foot is moved are paralyzed; in this case the deformity is usually in the form of a talipes equinovarus and cavus. Much more frequently talipes equinus

is caused by a partial paralysis, the flexors of the toes becoming contractured in consequence of paralysis of their antagonists. A rare form of pure equinus results from a paralysis of all the elevator muscles of the foot, the depressors remaining normal. Much more frequently we meet even in this case with equinovarus, resulting from a paralysis of all the muscles which flex the foot (the tibialis anticus, extensor longus hallicis, and extensor communis digitorum), and of the peronei, which extend the foot and force it into a condition of valgus; then the muscles of the calf alone remain active, and these not only depress the anterior portion of the foot but also produce varus. This latter condition may be still further increased if, as quite frequently happens, some power remains in the tibialis anticus as well as in the muscles of the calf, and only the extensor hallicis, extensor communis digitorum, and peronei are paralyzed. Finally, a condition of equinovarus may possibly also result if all the rest of the muscles of the leg are paralyzed, while the tibialis anticus is only in so far weakened that it is unable to antagonize the weight of the foot, but may still pull it over into a varus position. The very rare talipes equinovalgus may most readily be formed when all the muscles which move the foot, except the peronei, are paralyzed, or if, with paralysis of the flexors of the foot, the peronei are weaker than the true calf muscles.

The arching of the foot in talipes equinus is probably always of a static nature.

Talipes calcaneus occurs as a rule in paralysis of the depressors of the anterior portion of the foot, when the elevators are normal. As the muscles of the calf are usually alone paralyzed while the peronei are normal in this case, these muscles convert the talipes calcaneus into a calcaneovalgus and increase the arching of the foot (pes calcaneovalgus excavatus).

The very rarely encountered pes calcaneovarus may occur if the tibialis anticus alone remains intact and strong enough to raise the foot and to bring it into the position of varus.

Talipes valgus may be produced by a paralysis of the tibialis anticus alone, talipes varus by a paralysis of the extensor communis digitorum alone.

In the knee, genu recurvatum is most frequently met with; its occurrence is to be hoped for in the paralysis of all the muscles moving the knee-joint, as then only is walking possible. It will of course occur more readily, though this is very rare, when the quadriceps remains intact and the flexors of the knee are paralyzed. An active flexion of the knee is also very rarely met with; it occurs when the extensor muscles alone are paralyzed.

In the hip we usually meet with a flail joint. However, abduction, outward rotation, and slight flexion of the thigh may be present if the adductors are paralyzed and the abductors and the ileopsoas remain intact; and on the other hand there will be adduction and also flexion if the adductors and the ileopsoas are intact, and the abductors, namely the glutei, medius and minimus, are paralyzed. In the first instance a luxatio infrapubica and in the latter a luxatio iliaca paralytica may eventually appear.

Marked scoliosis or kyphoscoliosis may be the sequel of a unilateral or bilateral paralysis of the muscles of the trunk. Scoliosis may, however, also follow the shortening or the lengthening of one of the lower limbs, and lordosis a permanent flexion at the hip-joint.

A paralytic luxation of the shoulder-joint makes its appearance when nearly all the muscles of the shoulder, but especially the deltoid, are paralyzed.

In the elbow-joint we do not meet with deformities, as passive extension and flexion of the joint are always possible if only the arm can be moved at the shoulder. In the hand, flexion of the fingers occurs most frequently in paralysis of the extensor muscles, rarely extension of the hand and of the fingers in paralysis of the flexor muscles. Deformities which are difficult of reduction do not, however, occur in the upper extremities.

The final stage of the disease consists in nearly every case in the development of permanent contractures and deformities, which latter again are essentially a sequel to permanent muscular paralysis and atrophy. The patient is by this time to a greater or less degree a cripple, but was formerly thought to enjoy, aside from this, full mental and bodily vigor. We are aware at the present time, however, that this is not so in all cases. Raymond was the first who made the observation that, following a spinal paralysis which has occurred in childhood, a progressive muscular atrophy may occur years afterwards, and many other observers, the first of whom was Seeligmüller, have confirmed this. One of us (Bruns) has seen two cases with the residuum of an infantile spinal paralysis in one leg and a beginning progressive muscular atrophy of the small muscles of the hand. In these cases the distance of the two disease foci from each other is especially noteworthy. They remind one of the frequent occurrence of classical epilepsy at the age of puberty following an acute encephalitis at an early age, and also of cases in which the engrafting of a multiple sclerosis on an acute disseminated or a focal myelitis has taken place. According to Heine a recurrence of the inflammation in an acute form is possible. This will be again referred to in the section on Acute Poliomyelitis of Adults.

It may be well to review briefly what we have said concerning the course of the disease. This consists of an initial stage whose duration is from a few hours to a few days, a period of the greatest and most extensive paralysis whose duration at the most is a few weeks, and then convalescence or the retrogression of the paralysis which may continue possibly into the ninth month; at the same time contractures and deformities make their appearance in the areas of the permanently paralyzed muscles, and these may of course continue to progress for years.

Death, which very rarely occurs, may be caused by a very wide extension of the inflammatory process, as post-mortem cases seem to teach, or by cerebral complications. Our own opinion as regards the possibility of perfect recovery has been stated above. The rule, at all events, to which there are hardly any exceptions, is a partial cure and a final atrophic paralysis with its sequelæ in one or more muscle areas. Exceedingly rare, comparatively, is the late grafting of a progressive muscular atrophy on an infantile spinal paralysis which has run its course.

It is hardly necessary at this day to say that in scarcely any other disease is the connection between the anatomical process and its results and the clinical symptoms so clear as it is in infantile spinal paralysis. We know that this disease consists, in the main, in a destruction of one or more portions of the gray matter of the anterior horns, a degeneration which must lead always, and under all conditions, to a flaccid and degenerative atrophic paralysis of the involved muscle areas. We know, furthermore, that the localization of this paralysis corresponds in the main with what we know at the present time of the localization of the muscle centres, of whole extremities and of individual muscles, in the anterior horns of the different segments of the spinal cord. We, finally, know that considerable destruction of the spinal cord is not capable of perfect repair, therefore permanent forms of paralysis will remain behind in nearly all cases.

It is not necessary that there should be any change in the symptomatology when the inflammatory lesions are not sharply confined to the anterior horns. Even an extensive participation of the lateral columns need not provoke spastic symptoms, particularly when the paralyzed muscles are atrophied through disease of the anterior horns. Pain seems to be present occasionally in the beginning. We may not be able to determine the presence of distinct anæsthesia at first, but slight forms may, of course, escape observation. A slight disturbance of the function of the bladder seems, however, to be present in the beginning.

An explanation of the initial convulsions which not infrequently occur occasioned the older authors much difficulty. They were attributed to the result of the fever, or to reflex influences of the diseased spinal cord on the brain. At the present time authors are probably unanimous in regarding a direct participation of the brain in the general infection as a probable cause of the convulsions in these cases. It is precisely the frequent occurrence of cerebral symptoms that furnishes a basis for the assumption that in infantile spinal paralysis we are dealing only apparently with a sharply defined disease. It seems to us in the highest degree probable—and we are not alone in this view—that we are dealing with one and the same disease, only affecting different localities, in spinal paralysis and in those cases of cerebral paralysis which Strümpell has designated poliencephalitis acuta infantum. As a rule, the disease of the brain in infantile spinal paralysis can be only slight and circumscribed, as permanent symptoms do not frequently remain behind. The combination of a cerebral and a spinal infantile paralysis is a classical rarity, although such a case has recently been described by Lamy. Redlich also found lesions in the medulla oblongata, in poliomyelitis acuta, and Medin frequently observed cerebral paralysis as well as spinal paralysis in children, during the epidemic which he described.

Diagnosis.

The symptomatology of infantile spinal paralysis is so characteristic and in general so uniform that the diagnosis must be considered an easy one, if only the main features are always kept in view. It seems unnecessary to repeat these here. It is of course impossible to make the diagnosis readily in the febrile or nervous (cerebral) stage, but it may be made with some assurance after the second or paralytic stage has been reached; and mistakes are hardly possible by the end of the second week when the paralysis has receded from a large area which was primarily affected, and when the reaction of degeneration has appeared in the still paralyzed muscles. Later on the atrophy and the deformities will at once lead us to a proper diagnosis, and this will be confirmed by the reaction of degeneration which persists for a long time in the paralyzed muscles. As Erb remarks, a doubt as to the diagnosis will occur chiefly in those cases which have fully run their course, when there is no longer any response to electrical stimulation of the atrophied muscles.

In the differential diagnosis of diseases of the spinal cord, hæmatomyelia engages our attention especially. It is true that the latter nearly always immediately follows an injury; the same may, however,

be said of infantile spinal paralysis. Hemorrhage of the spinal cord affects by preference the gray matter, and may therefore sometimes perfectly simulate an infantile spinal paralysis if it is confined to the anterior horns; but as it usually destroys the posterior horns also, and sometimes even these alone, we nearly always find marked disturbances of sensibility. More frequently the symptoms of hæmatomyelia resemble those of syringomyelia.

Infantile spinal paralysis is, as we have seen, a myelitis of the gray matter of the anterior horns, but it does not sharply confine itself by any means to this area. As already mentioned, we find in it all degrees of extension, even a marked transverse myelitis, for example, a disseminated myelitis or a central diffuse myelitis. It is therefore possible to be in doubt occasionally whether to designate the case as myelitis or as poliomyelitis. If, however, we reserve the name myelitis for the transverse forms, the distinction will not be difficult; myelitis will then be distinguished by disturbance of the sense of feeling, disturbances of the function of the bladder, decubitus, usually also by spastic symptoms and increased reflexes. Myelitis moreover occurs very rarely in early infancy.

In caries of the spine and in tumors of the latter and of the meninges of the spinal cord, paralysis may sometimes occur suddenly. In this case, however, the picture presented will be that of a transverse lesion, and not of an atrophic paralysis of individual limbs or parts of limbs. In all these cases there will also usually have existed violent pain for a long time, and in disease of the spine frequently also deformities of the latter.

Progressive muscular atrophy, which may also occur in children, generally in a familial form, and progressive muscular dystrophy need only be mentioned to point out their distinguishing characteristics; both diseases are slow in their beginning and are progressive.

Infantile cerebral paralysis was formerly often mistaken for the spinal form, but at the present time this is hardly possible. The former occurs most frequently in a hemiplegic form, in which the face is nearly always implicated. Marked atrophy of the muscles is absent, although occasionally the only symptom remaining of former cerebral disease in childhood may be an imperfect development of the muscles and bones; the reaction of degeneration is absent. The paralysis is of a spastic nature, the reflexes are increased, frequently to the point of clonus; often involuntary muscular movements are present, especially athetosis, hemitremor, or hemichorea; the intellect may be impaired. Not only are convulsions present in the initial stage, but very often total or partial epilepsy is developed later

on. Of possible contractures, we meet with only the cerebral contractures of the hand and the fingers, and talipes equinus.

The cerebral diplegias might much more easily be confounded at the first glance with infantile spinal paralysis than the hemiplegias. The differences, however, in the nature of the paralysis and in the electrical conditions as compared with poliomyelitis anterior hold good here as well as in the hemiplegias. The spasmodic movements in these cases are especially frequent and marked, and the intelligence is nearly always affected. To this class belongs also what was formerly called spastic spinal paraplegia of childhood, and which is undoubtedly dependent in most cases upon some disease of the brain. It stands to reason that the diagnosis may become very difficult when a cerebral and a spinal paralysis are combined, but a careful examination should clear up the diagnosis.

Multiple neuritis in its beginning may easily be mistaken for poliomyelitis. But its course is different, its development is nearly always slow, with exacerbations and remissions, occasionally with true relapses. Convalescence is more uniform; all the muscles regain their functions slowly, but not all of them of course within exactly the same period nor in the same manner as in poliomyelitis, in which a large part of the muscular apparatus recovers during the first few weeks, the remainder, however, remaining paralyzed for a long time. Finally, a perfect cure nearly always obtains in neuritis, but scarcely ever in poliomyelitis. In neuritis violent pain also is nearly always present, and frequently the muscles and nerves are painful on pressure. As a rule, we do not find as typical a reaction of degeneration in neuritis as in poliomyelitis. The localization of the paralysis is not essentially different.

The typical form of obstetrical paralysis is that of Erb's plexus paresis, namely, paralysis and atrophy of the deltoid, infraspinatus, biceps, brachialis anticus, and supinator longus; it very rarely involves also the lower portion of the brachial plexus, or the latter only. This same grouping of the paralysis may also be found in infantile spinal paralysis. In this case the previous history is essential to the diagnosis. In affections of the lower portion of the brachial plexus an involvement of the sympathetic nerve—contraction of the pupil and narrowing of the fissure of the lids of the same side—would at once decide in favor of an obstetrical paralysis. Poliomyelitis is furthermore rare in the new-born.

The neural progressive muscular atrophy of Hoffmann may occasion paralysis, atrophy, and deformities of the lower limbs, but this is also a progressive affection, and involves sensation. It is besides of a familial nature.

Hysteria may at first sight present symptoms resembling those of infantile paralysis. Contractures of long duration of the ankles, causing equinus or calcaneus positions of the foot, are particularly liable to occur. Along with this the muscles of the leg may also be slightly atrophied. An electrical examination is usually sufficient for the purpose of diagnosis, but this must sometimes be made under an anæsthetic, owing to the great tension of hysterical muscular contracture; in most cases also the effect of the anæsthetic will be to relax the contraction, thus demonstrating that the deformity is not a fixed one. At the same time there may be present in hysterical contractures of long duration an actual shortening of the tendons and ligaments and finally changes within the joints.

The so-called syphilitic pseudoparalysis of the new-born of Parrot, which consists in an inability to flex the arm at the shoulder, on account of pain caused by the syphilitic disease of the diaphyses and epiphyses of the arm, may be mistaken for infantile paralysis, but it is more likely to be confounded with obstetrical paralysis. An electrical examination is not always decisive in these cases, as we know through Westphal that phenomena reminding one of the reaction of degeneration may occur in perfectly healthy new-born children. As a rule rapid recovery results in these cases on the exhibition of mercury or iodide of potassium.

In Barlow's disease—infantile scorbutus—we often meet with pseudoparalysis, especially of the lower extremities. These are, like those of Parrot's disease, occasioned by the pain produced in their movement, as infantile scorbutus has a predilection for the epiphyses and joints, and leads sometimes to separation of the epiphysis. The condition of the gums, the profound anæmia of the child, the continued fever, and the disturbances of digestion are characteristic symptoms of this disease. One of the writers met with extensive obviously hysterical anæsthesia a short time ago, in a case of infantile scurvy, which had led to the diagnosis of a myelitis. As Barlow's disease is usually met with in older children, the electrical examination will decide the issue here.

Hip disease, congenital dislocation of the hip, or severe rachitis would be liable to be mistaken for infantile spinal paralysis only on very superficial observation. Congenital clubfoot is perhaps due sometimes to a fœtal poliomyelitis; it may certainly occasionally be referred to developmental defect, namely, a myelomeningocele accompanied by spina bifida.

The diagnosis should not confine itself solely to the recognition of the infantile spinal paralysis, but we should endeavor as soon as possible to find out which of the muscles are paralyzed, this being of

the greatest importance in prognosis as well as in treatment. Generally this examination will first be made some time after the paralysis has occurred, and when the latter has already narrowed itself down in some measure to the areas in which permanent changes will be left. The demonstration of this paralysis, as in the adult who can be ordered to execute certain movements, is of course not often possible in children, but we may avail ourselves of the fact that when the extremity is immobilized but the joint to be examined remains free, and irritation is induced, say by pricking the limb with a needle, the limb will then usually, in the endeavor to escape the irritation, carry out all movements which it is possible for it to make; the seat and extent of the paralysis may then be determined from these defective movements. The most certain method, however, is the electrical examination; we may thus, after the lapse of a few weeks, determine, even in muscles which are still paralyzed, whether functional activity will return or not. This point has already been dwelt upon at some length above. Muscles showing the reaction of degeneration remain permanently paralyzed, those showing a weakened reaction will again recover. An accurate electrical examination, however, in these crying and actively resisting children who keep their normal muscles in continual motion is by no means easy of execution; the small size of these little patients and the large amount of subcutaneous adipose tissue also render the examination a difficult one, and not infrequently ignorant parents themselves put an end to what they regard as the excessive zeal of the physician. In the foot especially the conditions are frequently so complicated that a satisfactory conclusion may not be reached even when repeated examinations have been made with the patient in perfect repose, but only an approximate conception of the extent of the muscular paralysis is obtained. Practically, however, this will generally prove satisfactory. We have already considered in what manner the atrophies and deformities may present a clew to the seat of the paralysis, and in how far they may be made use of for this purpose.

Prognosis.

In respect to the prognosis of infantile spinal paralysis the following two propositions may be stated: (1) The prognosis as regards life is very favorable; (2) the prognosis as regards perfect recovery is very unfavorable.

Of the first it may be said that death as a result of infantile spinal paralysis is of rare occurrence. It may be, however, that it happens more frequently than we have imagined up to the present time, for it

is impossible for us to know whether some fatal cases of convulsions may not be due to a poliomyelitis acuta. This leads us at the same time to the remark that probably in most cases of a lethal outcome grave cerebral complications are present. It may be possible of course that a widely diffused poliomyelitis may of itself cause death, when it attacks for example the phrenic nerve. After the initial stage is passed, we need not, generally speaking, fear a lethal outcome.

As to the second proposition, the termination in a perfect cure is so very rare that it may practically be left out of consideration. A permanent paralysis is nearly always sure to remain in one or more extremities. We are very soon able to convince ourselves, by the electrical examination, whether one or more of these paralytic foci will remain permanently and what their location will be. The prognosis will then of course depend on the number and extent of the lesions, and also on their seat, in so far as the prognosis is, as a rule, more unfavorable in paralysis of an arm than in that of a leg, or of one of its parts. We must of course also take into consideration here the conditions of the individual segments of the limb, as well as the fact whether a total or only a partial paralysis of a joint is present. For on this, above all, will depend the occurrence and the kind of deformity, whose prognostic importance is naturally very great. Over against these unfavorable prognostic conditions, we may call especial attention to the fact that we are able in nearly all cases of extensive paralysis to foretell marked improvement, and with certainty if the electrical examination has previously given results of prognostic value. Poliomyelitis does not seem in most cases to exert any influence on the general health and on the later life of the patient. The occurrence in later years, however, of progressive spinal muscular atrophy, which has been not so infrequently observed, should induce us to be somewhat guarded in our prognosis.

Treatment.

The treatment of infantile spinal paralysis must of necessity be divided into that of the initial stage, the paralytic stage, and the sequelæ, and of course varies in each of these stages. In the last stage it is limited to the treatment of the deformities, and is then surgical rather than medical.

In the initial stage, the treatment is usually confined to the application of the general principles which guide us in the management of all febrile affections. Rest in bed is of course necessary, likewise a non-irritating, light diet—the bowels being regulated when necessary. More cannot be done, if only for the reason that it is impossible in

this stage to make a positive diagnosis, apart also from the fact that the physician is generally not called at this time. The probable presence of convulsions even cannot furnish ground for positive treatment. Should stupor appear, cold affusions may be applied while the child is in the warm bath. If there is difficult dentition at the time, the gum may be lanced. Even when paralysis is plainly present, and a correct diagnosis is thus rendered probable, we would advise against all active treatment, and that the child be disturbed as little as possible. All movement is to be avoided, also as far as possible loud crying, coughing, etc., should be restrained. In particular must we caution against the abstraction of blood by leeches or wet-cupping, or against a too energetic so-called derivation to the intestine or the skin. The favorable influence of these measures is very problematical, and that most of them act in a debilitating manner is certain. The diaphoretic treatment by wet packs, strongly recommended by Oppenheim, is unquestionably harmless, and may perhaps be of some benefit.

The electrical treatment of infantile spinal paralysis until recently enjoyed great repute, and even a directly curative influence was attributed to it. At the present time we know that this is impossible, for whatever nerve tissue has actually been destroyed by the anatomical process cannot be cured by the cerebral or peripheral application of electricity. The so-called central galvanization of the spinal cord, which was in former times recommended by most authors, is probably useless. As regards the peripheral application of electricity we may proceed as follows: In the first place it should not be applied too early, not until the greater part of the primary paralysis has receded, and not until it has been positively determined by an electrical examination which of the paralyzed muscles will recover their function and which will remain permanently paralyzed. The electrical treatment is then to be confined as far as possible to the former, for treatment of the muscles which show the reaction of degeneration is unnecessary, and perhaps even injurious. As the muscles, which will recover their function later, show only a quantitative lessening of their irritability, and are responsive to both the faradic and the galvanic current, it is really only a matter of personal preference which form of electricity we employ. Perhaps it may be even more correct, in opposition to the view formerly entertained, to choose the interrupted current, because this does not affect muscles showing the degeneration reaction. It is sufficient to employ faradization every second day. This treatment can at all events do no harm, and the possibility of a favorable influence on the nutrition of the muscles cannot be denied.

Massage may also be employed in place of electricity, or it may be practised alternately with it. In the beginning only light manipulations should be employed, but later kneading of the muscles may be resorted to. Actual gymnastic exercises and Swedish movements can be carried out only with older children.

Besides these methods, general tonic treatment may also be taken into consideration. This includes an outdoor life, nourishing food, baths, mud baths, mineral baths in indifferent or chloride of sodium waters, and other hydrotherapeutic methods. Baths and water cures should, however, not be employed too early. Cod-liver oil is also to be highly recommended under certain conditions.

It is also of the greatest importance to counteract from the very beginning the occurrence of contractures and deformities. We are convinced from personal experience in isolated but unfortunately very few cases, that a great number of contractures and deformities can be avoided by the use of very simple methods during the period of convalescence, which after they have once been established can either not be corrected at all or only by surgical measures. An important measure, and in this we agree with Oppenheim as against Seeligmüller, is to allow the children to lie abed as long as may be possible. Especially may a weak back and the deformities of the spine be totally avoided in this manner. The deformities of the lower extremities, particularly of the foot, are, as we have seen, undoubtedly due in part to early attempts at walking, which lead to a faulty position of the leg. Rest in bed alone is, however, not sufficient, but along with it various, but generally quite simple, methods are necessary for the prevention of crippling.

Let us consider the foot for the present. Here our measures must be applied to secure as far as possible the maintenance of the foot in a position at a right angle to the leg. Should a flail joint be present from paralysis of all the muscles of the foot we must prevent the pressure of the bed-clothing from forcing the foot into the position of equinus by protecting the limb with a wire cage. The sinking of the foot into the position of equinovarus by its own weight may in this case be avoided by keeping the foot at a right angle to the leg by means of a strip of adhesive plaster. If only a few of the muscles which are concerned in flexion and extension of the ankle joint are paralyzed, so that the active contracture of the non-paralyzed muscles is the main cause of the existing deformities, these measures are insufficient, and the foot must be put up in a side splint, Volkmann's splint, or a gutta-percha splint which is directly moulded to the foot, and only removed when the child is to be bathed, or electrically treated, or is to receive massage.

The contractures of the hip-joint (we now speak of flexion and abduction) are best avoided by extension with weights. So also the very rare cases of contracture of the flexors of the knee-joint. In extensive paralysis of these joints, deformities will not occur when the recumbent position is maintained.

Contractures of the flexors and extensors of the hand are also obviated by means of suitable splints. These need not be worn continuously, however, as the contractures are not usually of high degree. The paralytic subluxations of the arm may also be prevented by the application of a suitable suspension apparatus. It stands to reason that the manipulation of the joints by electricity, gymnastics, and massage also acts in a manner antagonistic to the contractures. After the patient is able to walk, the protective splints for the avoidance of deformities must, of course, be made stronger—slanting soles, for the avoidance of the varus or valgus deformities, and firm shoe splints, perhaps with a contrivance to raise or lower the toes. It is particularly important to prevent in time the appearance of a scoliosis, which is apt to occur through the shortening of a leg, by using a thick sole on the shoe of that foot.

We must confess to our sorrow that a knowledge of the importance, effectiveness, and easy execution of the prophylactic treatment of the deformities and contractures of infantile spinal paralysis exists almost as little among physicians as among the laity. Usually we are called on for treatment only in a late stage of convalescence when well-marked contractures already exist, or even later, and these cases are often seen by the orthopedic surgeon in a fearfully neglected condition. In this stage no benefit can be hoped for from either electrical or medicinal treatment, and the case is one for the orthopedic surgeon, since the treatment of the sequelæ of infantile spinal paralysis is a purely surgical one.

We cannot enter here into the details of orthopedic treatment, and will dwell only on a few general points and for the rest refer to the text-books on orthopedic surgery. Orthopedic measures may be divided into the use of mechanical appliances, such as bandages, elastic traction apparatus, and firm supporting splints, and true surgical operations, either minor operations which will finally make possible the use of apparatus, or major operations which will make the use of mechanical appliances altogether unnecessary. As a general rule, we endeavor nowadays to make the apparatus as simple as possible, or else, especially in those cases in which a perfect result may not be attained, to resort at once to operative measures. If no permanent deformities have appeared, all those forms of apparatus may first be employed which have been recommended above for the

prophylaxis of deformities, at the time when the patient again begins to use his legs; namely, firm splint shoes with perhaps elastic traction in the desired direction applied to the point of the foot, slanting soles, high cork soles for the feet, firm retention splints for the knee, to prevent especially too great a genu recurvatum; supporting apparatus for the hip-joint, and similar supports for the upper extremities. Most frequently, however, such a simple correction of the faulty position is not possible, on account of the shortening of the non-paralyzed continuously contracted muscles and their tendons, especially the muscles attached to the tendo Achillis. Surgical measures, either so-called *redressement forcé* or else a division of the tendo Achillis, must precede the correction of the deformity in such cases. After this has been accomplished, the foot is put up in a plaster-of-Paris dressing for some time, the deformity being at first over-corrected. Later a suitable retention splint is applied and passive motions are made regularly.

The division of the flexor tendons at the knee-joint will probably rarely be necessary. It would be well to apply extension by weights and to follow it up later with the use of a splint.

Old contractures at the hip-joint, especially if they are accompanied by a paralytic luxation of the head of the femur, can probably be rectified as a rule only after a division of the non-paralyzed and contractured muscles. In this manner alone a reposition of the luxated head of the femur can generally be accomplished. This would be best followed by extension with weights.

In very severe cases of clubfoot tenotomy alone is insufficient, and recourse must be had to resection of the bones of the foot.

Good results have in late years been obtained in infantile spinal paralysis by a new surgical procedure, which has been named arthrodesis by its author, Albert. This measure is to be especially considered in the worst forms of infantile spinal paralysis, in which a flail joint has resulted from a paralysis of nearly all the muscles of the joint. It consists, briefly, in producing an artificial ankylosis of the joint involved. It is especially applicable in a loose ankle-joint caused by a paralysis of all the muscles, also in non-rigid pes calcaneus and in a loose knee-joint. By this operation on the hip-joint we may frequently again obtain a useful limb, when only the ileo-psoas muscle is still active, and thereby avoid the expensive splints needing constant repair. We should avoid arthrodesis if possible, if only for the reason that sitting is nearly always made almost impossible by it, but it is sometimes necessary in paralytic luxation. In the arm arthrodesis will probably not be of much use when there is merely subluxation; on the other hand, it may, under some circum-

stances, be very useful for immobilizing the completely relaxed elbow-joint in a rectangular position.

We need only mention the so-called tendon transplantation. This consists in suturing the central end of the tendon of a non-paralyzed muscle of little importance to the peripheral end of the tendon of a paralyzed muscle whose function is important, and in this manner, so to speak, the function of one is transmitted to the other. This operation has been performed a number of times recently; the central end of the non-paralyzed extensor hallicis, for example, having been sutured to the peripheral end of the paralyzed extensor communis digitorum pedis or tibialis anticus; or in talipes calcaneus due to paralysis of the gastrocnemius the inner portion of the tendons of the flexor longus digitorum and peroneus has been joined to the tendo Achillis. Whether this method, which is ingenious, will be useful, time only can show; it is, at any rate, applicable only to a limited number of cases. To obtain the best results, it would probably be necessary to resort to this method as early as possible.

Poliomyelitis Anterior Acuta Adultorum.

Synonym.—Acute atrophic spinal paralysis (Westphal).

History.

M. Meyer seems to have been the first to have published a detailed clinical description of acute atrophic spinal paralysis of the adult. Duchenne, of Boulogne, eleven years later, described the same affection, and was the first to call especial attention to the perfect analogy of infantile spinal paralysis with that of the adult form. This view was then concurred in by a number of other observers. Although the question was originally one of clinical observation only, Gombault in 1873, and F. Schultze in 1878, demonstrated that the anatomical changes were the same as in infantile spinal paralysis, and that we had to deal with an acute myelitis confined chiefly to the gray matter of the anterior horns. This established fact could not be denied, even when it was discovered later that a great number of cases which had previously been classed with atrophic spinal paralysis were without doubt cases of true peripheral neuritis, which latter disease may present symptoms very similar to those of atrophic spinal paralysis. We must admit, however, that there exists in the adult a disease which is wholly analogous to infantile spinal paralysis. This poliomyelitis adultorum is, however, in itself, and especially in comparison with infantile spinal paralysis, a very rare disease. By reason of the close relation of poliomyelitis adultorum

with infantile spinal paralysis, it is needless to give a detailed description of this disease in all its parts. It will therefore suffice to present a general review, dwelling mostly on those few points in which the two diseases differ from each other.

Pathological Anatomy.

As previously stated, Gombault and F. Schultze demonstrated that we have to deal in poliomyelitis acuta adultorum as well as in infantile spinal paralysis with an acute myelitis of the gray matter of the anterior horns. Later observations, which have been few, have confirmed this. The inflammatory process of the spinal cord is not, however, confined to the anterior horns in the paralysis of adults, any more than it is in that of children, and though we are not yet in possession of facts which positively demonstrate this point, still it may be said with certainty that the inflammatory process in acute poliomyelitis of adults is confined mainly to the area supplied by the central artery.

Histological differences in these two forms of poliomyelitis do not exist. On the other hand, it would appear that the disease is usually more intense in the adult, in so far as in the adult a greater number of definitive lesions may remain in both the cervical and the lumbar enlargements, while in children frequently only one or two definite foci will remain.

Etiology.

It may also be stated as a fact that we have to deal with an infectious disease in acute spinal paralysis of adults as well as in that of children. This fact is even more marked here than in the children's disease, for while in the latter the affection possesses as a general rule the characteristics of a disease, more especially of an infection *sui generis*, and its occurrence as a complication or sequel of some recognized infectious disease is at all events very rare, the latter is nearly always the rule in poliomyelitis adultorum.

I have myself seen two characteristic cases following typhoid fever; it has also been observed after measles, in gonorrhœa (Oppenheim), in the puerperal state, etc. We may perhaps take for granted that *every* infection may, under certain conditions, give rise to these symptoms. Nevertheless, the occurrence of the disease as a primary affection has not infrequently been observed; or it might perhaps be more correct to say, the disease occurs in consequence of an infection by agents as yet unknown to us (*infections innommînées* of Marie). Heavy colds, traumatisms, and overexertion are also looked upon as

causes. These factors, however, act probably only along with the infection itself as auxiliary or predisposing causes.

According to some authorities, patients who have passed through an infantile paralysis are in later years more liable to suffer again from an acute poliomyelitis.

As regards the particular mode of infection, the reader is referred to the section on infantile paralysis. We will only mention that the disease, at least in its acute stage, corresponding to the character of the infection, does not limit itself to the spinal cord, but may implicate the entire economy, as is shown by the clinical symptoms. We have here another point of resemblance between atrophic spinal paralysis of the adult and infantile paralysis.

Symptoms.

The initial stage of atrophic spinal paralysis is always accompanied by fever. If the disease is a sequel to some other infectious disease which has run its course, or occurs seemingly as an independent affection, the advent of the fever will occur suddenly in the midst of perfect health; should it, however, appear as a complication of typhoid fever, for example, an additional rise of temperature may at the most be observed. The fever may continue in the adult for several weeks, while it lasts in children at the most three to four days. Initial cerebral symptoms, particularly convulsions, are rare in the adult, although stupor, coma, and delirium may sometimes be observed. This is probably due to the fact that in young children every irritation of the brain is liable to react with convulsions.

The occurrence of paralysis is usually rapid in the adult, as in the child; but the grown patient is naturally more observant than the child or its parents, and notices that at first one leg, then the other, and lastly the trunk and upper extremities are involved. In a short period of time the greater number of the muscles of the body are totally paralyzed. Adult patients complain much more frequently than children of intense radiating pain at the onset of paralysis, a fact which is easily explained by the extension of the anatomical process to the posterior columns and posterior horns; it is probable that these pains would be noted much more frequently in children also if the latter could make themselves better understood. Should the pain be very severe and lasting, a neuritis might of course be suspected. Disturbances of sensation, paralysis of the sphincters, and trophic disturbances of the skin are not encountered.

Death may take place during the first stage of the disease from an involvement of the motor centres of the muscles of respiration; this, however, as in infantile paralysis, occurs very rarely.

The subsequent course of the disease is the same as in infantile paralysis. The general health, after the fever has run its course, remains permanently good. The extent of the paralysis attains its maximum in the first few days of the disease, after which time it is not progressive but retrogressive.

In a relatively short period, a few weeks or months, a large portion of the paralyzed muscles again recover. Those which for the present remain paralyzed show either a complete reaction of degeneration, and then rapidly become permanently atrophic, or they show a diminution of electrical irritability with both currents; the latter may eventually recover, but very slowly and gradually, more slowly even than in infantile paralysis, so that we may still hope for improvement even after a very long period has elapsed. When paralysis occurs, it is of the relaxed variety. Finally a total absence of electrical irritability takes place, and all reflexes are abolished. As a general rule, electrical irritability is more slowly recovered than functional use.

As has already been said, it is the rule to have a greater number of permanent lesions in the spinal paralysis of adults than in that of children. Corresponding to this, several or all of the extremities are involved in the permanent paralysis. Of two cases observed by the writer (Bruns), there remained behind, in the first, a paralysis of all four extremities; in the second, of both arms and of the muscles in the area of the upper cervical cord, so that even the diaphragm was paralyzed. This same fact is mentioned by nearly all authors. The combinations of paralysis in the individual segments of the extremities do not vary from what we have seen in infantile spinal paralysis. The types of Remak also occur here. A permanent affection of the upper cervical region, with paralysis of the diaphragm, seems to occur more frequently in the spinal paralysis of adults than in that of children.

It was believed, before multiple neuritis was as yet well recognized, that acute atrophic spinal paralysis frequently resulted in perfect recovery. At the present time we know that this is as infrequent, if it happens at all, as in infantile spinal paralysis. A permanent paralysis is nearly always left behind in one, or more generally in several extremities. The termination of a case in perfect recovery necessitates, according to our view, a diagnosis of multiple neuritis instead of that of poliomyelitis anterior.

An essential difference between infantile poliomyelitis and that of the adult consists in this, that the deformities so frequently occurring in the one do not play an important rôle in the other. In the first place, all those deformities which are occasioned in infantile pa-

ralysis by the disturbance of osseous growth do not occur, as this has already reached its close. In the second place, the attention of the adult is of course at once drawn to any beginning contractures and faulty positions, and he avoids the injurious factors mechanically active in their production, and is thus able to arrest their further progress. This is also a hint to us that we might be able to prevent these deformities in children if we would devote to this object the necessary care and attention. Deformities in adults are, of course, to be avoided altogether only by the use of a proper apparatus.

Diagnosis.

The diagnosis of acute atrophic spinal paralysis of the adult would be easy, with its specific symptoms, if it were always possible to differentiate it from neuritis. This is, however, often exceedingly difficult. An acute beginning with rapid progress of the paralysis to its highest point, severe pain, a convalescence at times rapid, at times slower, atrophy, reaction of degeneration, and a flaccid type of paralysis, are found in both diseases. Combined paralyses similar to those of poliomyelitis may also occur in neuritis. The occurrence of the disease following infectious diseases also is as characteristic of neuritis as of poliomyelitis. All these factors frequently render a differentiation impossible in the beginning, and a positive diagnosis can be based only upon the outcome—a perfect cure in neuritis, a partial one only in poliomyelitis. In doubtful cases we may bear in mind that multiple neuritis is a much more frequent disease than poliomyelitis adultorum. Another difficulty arises from the fact that the two diseases may coexist, and we now know that even in true multiple neuritis there is not a perfectly normal condition of the spinal cord.

It might be perhaps possible to hazard a diagnosis of poliomyelitis at the outset of the disease, if the paralysis has come on very suddenly, more so than it usually does in neuritis, and if pain is absolutely absent. The reaction of degeneration also is usually more typical in spinal paralysis than in neuritis. In a prognostic sense, the early differential diagnosis is of course of great importance.

Hæmatomyelitis is differentiated from acute poliomyelitis of adults by its course, which is rather more rapid than that of poliomyelitis, and by the absence of sensory disturbances.

Landry's paralysis is diagnosed by its course, and also by the absence, in typical cases of the latter, of the reaction of degeneration.

Prognosis.

The prognosis of acute poliomyelitis of the adult is more favorable in one respect than that of the infantile form of the disease, because the occurrence of deformities need hardly be feared; but in another respect it is less favorable, because the permanent paralysis is much more diffuse. The prognosis is always grave; although death rarely results, the patient usually remains a cripple and unable to support himself.

Treatment.

The therapy of the disease is the same as that of infantile paralysis. In the very chronic stage of convalescence, a sojourn at one of the indifferent or saline mineral springs is to be strongly recommended. In cases of very severe paralysis, apparatus and bandages for support, possibly also arthrodesis, must be employed. In general, however, surgery is of much less benefit in the atrophic spinal paralysis of adults than in that of infants.

Poliomyelitis Anterior Subacuta.

This affection was described by Duchenne under the name of paralysie spinale antérieure subaiguë. It is a more or less extensive atrophic paralysis, due to an inflammation of the gray matter of the anterior horns, which reaches its full development in the course of a few weeks. The frequency of its occurrence was formerly greatly overestimated, for, as we now know, most of the cases included here were really instances of multiple neuritis. Other cases, which were formerly described under this name, and in which anatomical research has furnished the proof of a disease of the spinal cord, do not belong clinically to this category, for their course was a progressively fatal one. But in subacute poliomyelitis the process must of necessity come finally to a standstill, and then more or less improvement in the paralysis will take place.

Subacute poliomyelitis anterior may differ in its *symptoms* from the acute form only in its slower development, paralysis of the muscles being always preceded by their atrophy. One muscle after the other, or one extremity after the other is involved in the paralytic process, an order indicative of the segment type of lesion.

In the *diagnosis* multiple neuritis alone is to be considered. As a general rule, we have to deal with the latter and not with poliomyelitis, certainly so in those cases which go on to perfect recovery.

The *prognosis* is not so favorable in these cases as in the acute, as we are necessarily for a long time in doubt whether the paralysis will come to a standstill at all, and if so, at what time.

Poliomyelitis Anterior Chronica.

The symptomatology of chronic atrophic spinal paralysis was considered doubtful for some time after the chronic forms of neuritis had been recognized, for the reason that the anatomical demonstration of its existence was so very defective. Oppenheim recorded an anatomically clear case of chronic atrophic spinal paralysis in 1888, and a second one in 1892. Recent authors have assumed that we have to deal, in this affection, with two forms of disease. First, with a real chronic inflammatory process originating in the blood-vessels, perfectly analogous to acute poliomyelitis anterior, only running a chronic course; and secondly, with a simple primary, perhaps a systemic degeneration of the ganglia of the anterior horns.

Pathological Anatomy.

Oppenheim's cases illustrate these two forms to which we have just referred. In the first case there were found a simple disappearance of the motor cells of the ganglia, moderate degeneration of the anterior roots, and more marked degeneration of the peripheral nerves, with resultant changes in the muscles, but no alterations in the blood-vessels; the process was confined to the anterior horns, and in the lateral columns to the processes of the cells of the lateral columns. In the second there was a notable increase in the number of vessels, the glia was softened, and numerous spider cells were found; the posterior columns and Clarke's columns were also slightly involved in the inflammatory process. The histological picture in this second case was entirely that of acute poliomyelitis.

Etiology.

Very little is known of the causes of chronic poliomyelitis. As quite similar affections have been observed to occur after lead-poisoning, it has been thought that there may be some connection with plumbism. Nonne has recently described a case of chronic atrophic paralysis occurring in diabetes. In all probability these cases of intoxication belong to the second type of Oppenheim's cases. Cases of primary progressive degeneration of the ganglia of the anterior horns are very obscure in their etiology.

Symptoms and Course.

The disease begins slowly, without any prodromic febrile stage; the patient perhaps becomes aware of a weakness in one or other part of one lower extremity, then another part becomes paretic, and later the trunk, the upper extremities, and finally the muscles of the head and neck are affected. More rarely the weakness is first experienced in the upper extremities. This weakness after a few months gradually increases in the muscular areas first attacked until it amounts to a paralysis with atrophy. During this time severe pain is frequently experienced, perhaps in those cases belonging to Oppenheim's second category more especially.

Examination at this time reveals a flaccid atrophic paralysis. The atrophy probably has followed the paralysis rapidly, but it never precedes it. The electrical reactions indicate degeneration. We shall find in the paralyzed muscles complete and partial reaction of degeneration, and frequently also electrical peculiarities, such as delayed indirect contraction or faradic reaction of degeneration. We occasionally find a normal reaction in the paralyzed muscles, with the reaction of degeneration in muscles which still retain their functions. The tendon reflexes are weak or abolished; they may be delayed, or may become exhausted after a few trials.

Disturbances of sensibility or of the sphincters are never observed. The reproductive function remains unaffected.

The segment type is not so marked in these as in the acute forms, probably on account of the very slow progress of the disease, but evidences of it may exist. Thus in the arm the triceps may for a long time remain unaffected; in the forearm, the flexor muscles of the fingers and the abductor pollicis; in the leg, the muscles of the calf or even the tibialis anticus.

The further course may now vary. In most cases the disease is a continually progressive one. More and more muscles are involved, and those affected grow constantly weaker, and then become atrophied. Not infrequently the disease takes on an ascending character, or it arises from several primary foci, from which it spreads eccentrically. Finally, there remains a total paralysis of all the extremities and of the trunk. If now the nuclei of the respiratory muscles also become involved, death by asphyxia is the result.

This course is probably inevitable in primary atrophy of the ganglia. The duration of the affection may then be about three years. In the non-inflammatory cases, however, there may occur a cessation of the process, with continuation of the paralysis in this stage, after

the former has advanced to a certain extent. Improvement here also is very rare. Should a cure take place in a suspected case, we may regard it in all probability as really one of neuritis. The patients are usually attacked in the prime of life, and men are apparently somewhat more exposed than are women.

Diagnosis.

The diagnosis of the disease rests upon the existence of a paralysis of the muscles and their degeneration. Its diagnosis may be easy in those cases which are characteristic in their symptoms and course. There is very little danger of confounding poliomyelitis with a truly chronic course with neuritis. Progressive spinal muscular atrophy probably does not differ greatly in its symptoms from those forms of chronic poliomyelitis which take on a progressive course. As its course, however, is also much slower, and one muscular fibre after another becomes degenerated, the clinical difference arises that while in poliomyelitis the paralysis precedes the atrophy, in progressive spinal atrophy the atrophy makes its appearance first, and paralysis can be demonstrated only after this atrophy has become more or less marked.

For this reason also the reaction of degeneration is rarely marked in progressive muscular atrophy, and frequently it can hardly be found. Furthermore, spinal muscular atrophy usually begins in the fingers and hands, chronic poliomyelitis in the legs. It goes without saying that forms are met with which run into each other, in which a positive distinction is not possible.

From amyotrophic lateral sclerosis, chronic atrophic spinal paralysis is differentiated by the fact that in it the paralysis is also in the legs a relaxed one from the beginning, and that the reflexes are absent or are weak; the atrophy begins in the hands in amyotrophic lateral sclerosis, and the bulbar symptoms also play a much greater rôle in it than in poliomyelitis anterior chronica. Syringomyelia differs so greatly in typical cases from poliomyelitis, by its trophic and sensory disturbances, that a mistake can scarcely be made. Only in those cases in which the disease is manifested principally by the paralysis and atrophy of muscles, and at the same time involves more rapidly than usual a whole extremity, must the diagnosis for a time remain in doubt.

Prognosis.

The prognosis in poliomyelitis anterior chronica is very grave. As a rule, the affection runs a progressive course to a lethal ending. We may well say with Oppenheim that the clearer the symptomatol-

ogy the worse is the prognosis. In cases in which there are violent pains the prognosis is less unfavorable, as pain is usually not met with in primary degenerative processes of the anterior horns; and it is in these latter cases especially that the paralysis progresses unchecked. When the pain is very marked, we may suspect a complication with neuritis. If this is the case, the progress of the affection may become arrested, and some amelioration even may occur.

Treatment.

This is wholly ineffectual in the continuously progressive cases. If the disease become stationary, it will not be due to our endeavors. Electricity, massage, and hydrotherapy may be tried, if the necessity for action is felt.

MYELITIS.

Under the general term myelitis, or inflammation of the spinal cord, we shall include a description of acute and chronic transverse myelitis, Landry's paralysis, and acute disseminated encephalomyelitis.

Definition.

In giving a description of so-called myelitis it is necessary—perhaps more so than in the case of any other disease—to obtain in advance a clear idea of what is to be included in this category and what is to be separated from it. The great difficulty of giving such a clear-cut definition of the clinical picture of myelitis is shown most clearly by the differences in the views of the most authoritative writers on the subject. Oppenheim, relying upon extensive clinical and anatomicopathological experience, looks upon acute myelitis as an extremely rare disease, whose diagnosis may be made only after the exclusion of all other possibilities. He believes that the nature of myelitis is more obscure than that of the majority of other spinal diseases, and he thinks that chronic myelitis depends, in most cases, on multiple sclerosis. On the other hand, Leyden, who includes in the category of myelitis all other chronic diseases of the spinal cord (with the exception of tabes dorsalis, progressive spinal muscular atrophy, Friedreich's disease, and syringomyelia), regards it as one of the best known diseases of the cord. Gowers also speaks of the frequency of acute myelitis and even of a special predisposition of the cord to inflammatory disease, but he includes many things in this category which it is better to keep distinct. Pierre Marie is very radical in upholding Oppenheim's views. He would prefer to drop

the term myelitis entirely from the nosology of the spinal cord and retains it for the present for clinical and didactic reasons only.

It might be supposed *a priori* that pathological anatomy would decide the question and that from its standpoint it would be easy to determine which diseases merit the term inflammation and which do not. But this is by no means true. The histological notion as well as the interpretation of the real nature of inflammation is still a variable one, and this is particularly true in regard to the spinal cord. It will be shown hereafter that the macroscopical and microscopical findings which are regarded as characteristic of acute myelitis are by no means characteristic of inflammation, but occur in exactly the same way in non-inflammatory diseases of the cord. Moreover, the finer pathological histology of the cord is still obscure. We need merely recall, for example, that we are at the very beginning of our knowledge of the ganglion cells. In this direction, accordingly, it is not possible to secure a satisfactory definition of the clinical picture of myelitis. We shall succeed better by basing our considerations on etiological conditions. We may include among the myelitides those diseases of the cord which, by their etiology and clinical symptoms, make it plausible or even indisputable that we have to deal with a virus which has passed through the blood-vessels to the cord and there exerted a destructive action. We have to deal here in the main with two large etiological groups: the infectious diseases and the intoxications in the narrower sense. Both groups may include a large number of diseases of the cord which are very different clinically, but are generally regarded to-day as inflammatory in character. They include acute and subacute anterior poliomyelitis of adults and children, the cerebral form of Landry's paralysis, those forms of combined system diseases which depend probably on a cortical myelitis, disseminated myelitis and encephalomyelitis (to which multiple sclerosis is undoubtedly closely allied), disease of the cord as the result of severe trophic disorders (pernicious anæmia) or the ingestion of poisoned articles of food (ergotism, pellagra, lathyrism), and finally those cases of more diffuse inflammation which spread irregularly over the entire transverse section of the organ and are known as transverse myelitis.

There are no essential differences between these various inflammatory diseases of the spinal cord. The special localization probably depends on the fact that in one case the inflammation-producing substance enters the territory of the central arteries (anterior poliomyelitis and some cases of Landry's paralysis), in another affects the entire central nervous system in a disseminated form (disseminated encephalomyelitis, multiple sclerosis), in a third series enters mainly

through the cortical vessels (certain forms of combined system disease), and finally, in a fourth group, affects the cortical and central vessels uniformly but only over a limited area longitudinally (transverse myelitis). Despite this close alliance the course of development of the doctrine of cord diseases has been such that almost all the diseases mentioned are discussed in separate chapters, for example, acute poliomyelitis of adults and children, multiple sclerosis, and the combined system diseases. This separation has borne abundant fruit for the development of our knowledge of the individual form of disease and for this reason we shall not deviate from it. In the main we shall discuss only the transverse form of myelitis, limited in extent longitudinally (transverse and more or less diffuse myelitis) and shall reserve for it alone the term myelitis. But included with it we shall also discuss Landry's form of myelitis (which in certain clinically rare forms can hardly be distinguished at the outset from transverse myelitis) and disseminated encephalomyelitis, which has been treated hitherto with scant courtesy and for which it is difficult to find another place.

If we confine the use of the term myelitis to the diseases which are characterized by their etiology as inflammatory and which attack the entire cross section of the cord over a varying extent longitudinally, we shall be convinced that myelitis is a rare disease, and that Oppenheim and Marie, not Leyden and Gowers, have advanced the correct view. Apart from the above-mentioned truly inflammatory diseases of the cord, which have been separated from myelitis on account of their special symptomatology and in a measure on account of historical traditions, the increase in our clinical and anatomicopathological knowledge has led us to separate from myelitis an entire series of diseases which were included, until recent times, in this category, and had swollen it to such an extent that it embraced the greater part of the diseases of the cord. Thus, the terms compression myelitis and traumatic myelitis were employed formerly, and indeed are in use even to-day, although it is known that true inflammation of the cord is rarely due to acute traumatic compression, to slow compression by tumors, or to compression from tuberculous disease of the vertebræ. It would be better to dispense entirely with the term traumatic and compression myelitis.

A large part of the cases of acute so-called myelitis are due to an acute thrombotic softening, not to an inflammation. These cases are entirely analogous to the long known thrombotic softenings of the brain. It might almost be said that the entire doctrine of acute myelitis, and particularly its pathological anatomy, has been erected upon such cases of vascular softening. Many cases of acute syphilitic mye-

litis in particular appear to develop in this manner, for it is well known that diseases of the vessels occur very extensively and early in syphilis. In such conditions we have to deal with the usually very acute syphilitic paraplegias, which often occur without any prodromes. In cases of meningomyelitis and in the rare true syphilitic inflammation which is confined to the cord, the entire process runs a slow course and the paralysis is usually preceded by other symptoms, especially pain. But a slowly progressive softening is not excluded, even when the cause is vascular, and hence the cases described by Erb as syphilitic spinal paralysis may be due in part to such causes, while in part they are residua of acutely developing paraplegias. Inasmuch as syphilis is not the sole cause of the vascular affections which lead to thrombosis, similar diseases of the cord will naturally occur under other conditions, but they are undoubtedly rare outside of syphilis.

Formerly syringomyelia was also regarded generally as a chronic myelitis, especially in those cases in which the trophic disturbances of the muscles are less prominent and the disease gives rise chiefly to paraplegic symptoms. On the other hand, the non-inflammatory nature of the secondary degenerations was early recognized. In like manner the very large group of hereditary and familial diseases of the cord, and which in the main are confined to certain systems, are hardly ever included among inflammations. The tabetic process also, at least so far as it concerns the spinal cord, is now recognized definitively as non-inflammatory. It is still questionable whether the diseases of the white columns in progressive paralysis do not depend upon a primary inflammatory disease of the cells of the gray substance, and Marie thinks that this is also possible in regard to amyotrophic lateral sclerosis.

Finally, the field of myelitis has been materially narrowed by the investigation of multiple neuritis. All the forms of this disease were regarded formerly as poliomyelitis or diffuse myelitis, while to-day we make the diagnosis of neuritis much more often than that of myelitis. Our enlarged knowledge of hysteria has also enabled us to recognize cases of this curable disease, when formerly the diagnosis of chronic myelitis would undoubtedly have been made.

If we take all these circumstances into consideration, we are forced to admit that we can rarely make the diagnosis of transverse myelitis with any degree of certainty, and that this morbid group has lost considerably in importance. Nevertheless myelitis still possesses practical and scientific importance. Its practical significance is based on the fact that we are compelled to make the diagnosis of myelitis quite often, at least provisionally, because the real cause of the para-

plegia, perhaps a tumor, is not recognized during life or only at a late period. The scientific significance of the small number of cases of true transverse myelitis resides in its etiology. In this particular we have made considerable progress in the last few years, and the knowledge we have gained is even calculated to throw new light on the pathology of other diseases of the nervous system.

History.

The first period of the history of myelitis extends from the beginnings of medicine to the epoch-making works of Ollivier d'Angers and Abercrombie, both of which appeared at about the same time in the first third of this century. Until that time diseases of the vertebræ, the membranes, and the spinal cord were thrown together without any clear distinction. These two authors, basing their studies upon a large number of clinical and anatomicopathological observations, first separated diseases of the spinal cord proper, and Ollivier employed the term myelitis which he had borrowed from Harless. The two writers made only macroscopic observations, but their findings were of the greatest importance at that time. In particular, they recognized the connection of acute myelitis with softening of the cord.

The second period extends from the time of these writers until the appearance of Leyden's text-book in the beginning of the seventies. The development of our knowledge of myelitis at this time depended mainly on our constantly increasing clinical experience and the careful histological examination of the normal and pathological cord. Aid was also furnished by physiological experimentation. It is impossible to give even approximately the names of all the pioneer authors. We will merely mention Dujardin-Beaumetz, Brown-Séquard, Hayem, and especially Charcot in France; Lockhart Clarke and Gull in England; Hasse, Engelke, Westphal, Fromann, and Manukopf in Germany. In 1875 Leyden collected the experiences of these writers and his own abundant observations in the principal work of his life, the "Klinik der Rückenmarkskrankheiten." To a certain extent he also marks the beginning of the third period, because he succeeded in excluding from the category of myelitis much that had formerly been included in it. This gradual restriction and finally marked narrowing of the clinical picture of myelitis, in accordance with the constantly advancing specialization of our knowledge, is the chief merit of the third period, at which we have now arrived. The manner in which this specialization was effected is evident in part from our definition of myelitis given above. Erb still calls myelitis the most important and extensive chapter in the doctrine of diseases

of the cord, but he is aware that much described under that heading is not inflammatory in the strict sense of the term. Pick differentiated syringomyelia and the large group of multiple neuritides from myelitis. The most effective and, at all events, the best prepared attack on the theory of myelitis was made by Oppenheim, seconded by Marie. To-day the clinical picture of myelitis, at least in the opinion of the majority of recent writers, exists only in the very limited manner which we have described above. It is in consequence of this that reports of cases of inflammation of the spinal cord have recently become very scanty. The main interest now centres in the etiology, and much has been achieved here by experimental investigation.

Until the time of Ollivier the history of so-called chronic transverse myelitis cannot be separated from that of the acute form. Later this was made to include, for a time, the findings of Türck and Rokitansky in regard to secondary degenerations. The clinical and pathological investigations of Charcot, Bourneville, Vulpian, and Ordenstein referred particularly to multiple sclerosis. The occurrence of chronic myelitis, apart from that which follows an acute inflammation, has been rendered very doubtful. Oppenheim believes, and we agree entirely with him, that we almost always have to deal with multiple sclerosis; Leyden, who also includes in this category the so-called combined system diseases, recognizes their more frequent occurrence.

Our knowledge of Landry's paralysis begins in 1859 with Landry's communication on acute ascending spinal paralysis without anatomical findings. The succeeding publications, although some of them mentioned organic findings, merely confirmed Landry's statements, and with Westphal's investigations in 1876, the essential characteristic of this disease, viz., the negative anatomical appearances, could be regarded as proven. But soon afterwards it was found that, while in some cases there was no disease of the cord, an affection of the peripheral nerves could be demonstrated; and the investigations of Oettinger and Marinesco—and even the early ones of Baumgarten and Eisenlohr—showed that very careful examination would sometimes reveal positive changes in the spinal cord. At the present time we distinguish a central and peripheral form of Landry's paralysis; the first variety will be discussed here.

The history of disseminated encephalomyelitis is of very recent date. The names most prominently associated with it are those of Westphal, Ebstein, Leyden, Küstner and Brosin.

Etiology.

The chapter on etiology is the most interesting and in many respects the most important in the whole doctrine of myelitis. We have already stated that, so far as regards acute transverse myelitis, Landry's paralysis, and disseminated encephalomyelitis, there are, in the main, two etiologial factors, viz., the infections and the intoxications. So far as regards infections, it may be asserted that the possibility of an implication of the spinal cord is present in every infectious disease, although some attack the cord more frequently than others. In the individual case the development of a myelitis will depend upon the fact whether—perhaps as a matter of accident—the amount of infectious matter which penetrates the cord is sufficient to produce disease or whether the cord is a *locus minoris resistentiæ* when the virus is present in the general circulation. The occurrence of acute transverse myelitis has been observed—in part only clinically—after measles, scarlatina, acute articular rheumatism, pneumonia, typhoid fever, malaria, diphtheria, and, especially in the last few years, after influenza. Recently the development of true myelitis after gonorrhœa has also been demonstrated (Leyden, Barrié). The occurrence of suppurative meningomyelitis in such cases has long been recognized.

Syphilis probably does not produce true inflammatory affections in an acute form. The gummous infiltration which penetrates the cord from the meninges or develops primarily in the former organ, will run at the most a subacute course. In the large majority of other syphilitic diseases of the cord we do not have to deal with inflammation. In the majority of cases of tuberculosis there is a simple compression of the cord as the result of the extradural carious processes. More rare are solitary tubercles in the cord itself, and still rarer is a true inflammatory, miliary infiltration of the cord in general miliary tuberculosis or secondary to a carious osseous focus. In tuberculous meningitis, on the other hand, small miliary nodules not infrequently penetrate the cord.

So far as regards wound infections, cases of transverse myelitis have been observed after erysiplas, septicæmia, anthrax, and, with special frequency, after peripheral infection. We are further compelled, for clinical reasons, to assume infection in an entire series of cases, although we are unable to make a definite statement concerning the nature of the infection-producer. Marie speaks of "infections innominées." This applies, for example, in the majority of cases of anterior poliomyelitis and not so infrequently in Landry's

paralysis. It also appears as if the bacillus coli communis may under certain circumstances produce myelitis, either directly or after the formation of a toxin.

Disseminated encephalomyelitis and Landry's paralysis occur after the same primary infections, and indeed the direct connection between the infection and myelitis is usually much more evident in these forms of disease than in transverse myelitis. Apart from the diseases mentioned above, encephalomyelitis has also been observed after cholera, cholera morbus, dysentery, and simple chronic diarrhœa. The first cases of this kind (after dysentery) were described by Gubler as early as 1860; then followed, in the seventies, the cases of Westphal after smallpox, of Ebstein after typhoïd fever; Küstner and Brosin saw a case which was probably connected with an old gonorrhœa, and Oppenheim mentions its occurrence in tuberculosis.

Landry's paralysis has been observed in direct connection with anthrax (Baumgarten), typhoid fever (Eisenlohr), smallpox (Oettinger and Marinesco).

A considerable increase in the extent and accuracy of our knowledge of the relation between infectious processes and myelitis has been acquired recently by experimental investigations. These we owe chiefly to French observers. Myelitis was produced by Babinski and Charrin by infection with bacillus pyocyaneus, by Roux and Yersin with the diphtheria bacillus and the bacillus coli communis, by Roger with the erysipelas streptococcus. Similar experiments were made by Gilbert, Manfredi and Traversi, Vincent, and Besançon. The similar action of the toxins has been proven in regard to diphtheria. Not alone transverse and disseminated myelitis may be produced in this way, but also partial forms of myelitis—partial in regard to the transverse section. Thus, the experiments of Babinski and Charrin resulted in a spastic symptom complex, while Roger produced a form similar to progressive muscular atrophy. Years ago the attempt was made (Hayem, Lionville, and Grancher) to produce myelitis by the direct action of chemicals, such as iodine and glycerin, upon the cord, but the majority of the results were not free from suspicion. The most successful of these experiments were made by Leyden with Fowler's solution.

The manner in which an infectious disease attacks the spinal cord may be threefold. In the first place the agents of the primary infection may penetrate directly into the cord; in the second place, secondary infections may be engrafted upon the primary infection, for example, upon a diphtheritic or typhoid ulcer, and the cord may be attacked by this secondary infection; thirdly, the toxins produced in the body by the primary disease may constitute the real morbific

element in the cord. The first method has been rarely observed. In one case of ascending paralysis Baumgarten found anthrax bacilli in the cord. In a similar case after typhoid fever Curschmann found typhoid bacilli, which he was able to propagate in pure cultures. According to Grasset, the second method is especially frequent. The primarily affected parts are usually colonized by the simple pus cocci, which penetrate thence into the general circulation and so reach the spinal cord. The infection seems to occur in this way in many cases after diphtheria. Eisenlohr also found staphylococci in the cord in a case of myelitis after typhoid fever. Oettinger and Marinesco found streptococci in ascending paralysis after smallpox, Barrié found staphylococci in a case of myelitis after gonorrhœa. Equally frequent, or perhaps still more frequent than this method, is the poisoning of the cord by toxins of the primary diseases. It is true that they have not yet been found in the cord, but that they may produce myelitis has been proven by Roux and Yersin in regard to the diphtheria toxin.

An inference in regard to the manner in which the infection has acted may perhaps be made from the clinical course of the disease in individual cases. The myelitis which is produced by the primary infectious agent would probably appear about the same time as the primary disease; the myelitis due to toxins might follow the primary disease after the lapse of weeks and months; an intermediate position, in this respect, would be held by cases in which the myelitis is due to secondary inflammation-producers. Whether a diffuse transverse myelitis, Landry's paralysis, or disseminated encephalomyelitis develops, will depend upon circumstances. In the first event the virus penetrates only a circumscribed part of the cord, but enters all the vessels of this region; in the second event the central vessels of the cord are chiefly affected; and in disseminated myelitis it extends over the entire vascular tract, but is scattered in different parts and penetrates the cord in small foci. Our anatomical knowledge of the distribution of the blood-vessels in the cord teaches us that the first and third conditions will develop much more readily than the second, and we therefore infer—as is really the case—that transverse myelitis will be rarer than the disseminated form, and perhaps even rarer than Landry's paralysis.

We must remember, however, that, according to the general belief, peripheral neuritis will develop much more frequently than myelitis after any of the above-mentioned infectious diseases. At all events the differential diagnosis between the two processes is a difficult one, and it has become the general custom to interpret a termination in recovery as favorable to the diagnosis of neuritis rather

than of myelitis. Recent careful investigations have shown us that in neuritis the cord rarely is entirely unaffected. Oppenheim believes that he is in a position to maintain that in those cases in which the symptoms indicate a combination of myelitis and neuritis, the prognosis is favorable also in regard to the myelitic process. But it is still certain that, on the whole, the processes which are confined to the peripheral nerves are more frequent than those resident in the cord, and that, other things being equal, they offer a much more favorable outlook for recovery. In regard to paralyses after intoxications there is a still greater tendency at the present time to attribute them to neuritic disease, and as a general thing this view is doubtless correct. But it is not very probable *a priori* that a virus which, when received into the economy, leads to paralyses, will injure only the peripheral nerves and not at all the central nervous system. In a certain number of toxic paralyses the implication of the cord in the shape of a myelitis has been demonstrated. Among the metallic poisons this has been proven positively in regard to lead and arsenic, but it is doubtful in regard to phosphorus and mercury. Among organic substances the most important in this respect is alcohol; in chronic alcoholism the centres as well as the nerves are injured. Diseases similar to acute myelitis also occur after poisoning with carbon sulphide. Gaseous poisons appear to have a predilection for the production of disseminated myelitis. This is proven in regard to carbonic oxide and carbon sulphide, and we have observed it also after chloroform anæsthesia. However, poisons, like infections, may give rise to transverse or disseminated myelitis or to ascending paralysis.

Vegetable poisons, as a general thing, appear to produce more chronic processes, and, in addition, have a greater tendency to attack individual parts of the cord than to cause a true transverse myelitis. This category includes ergotism and pellagra, with combined system diseases, and probably also lathyrism, whose anatomical basis is still unknown. Among animal poisons snake virus seems to be capable of producing myelitis. This category also includes rabies and the myelitis occurring after the ingestion of spoiled articles of food, particularly sausage poisoning.

Finally, in certain diseases toxins may develop in the organism itself and may give rise to myelitis. Thus a poliomyelitis was observed by Nonné in diabetes, and Oppenheim describes a transverse myelitis in tuberculosis and in the cachexia of cancer. This category probably includes the cases of myelitis occurring in pernicious anæmia, icterus gravis, Addison's disease, and after intoxications starting from the stomach and intestines. The bacillus coli communis perhaps plays a part in the latter cases.

Chronic myelitis usually develops out of the acute form; an acute stage is either followed by a slowly progressive inflammation, or the latter is interrupted occasionally by acute exacerbations. The latter course is especially characteristic of multiple sclerosis.

Although the infections and intoxications play by far the most important rôle in the genesis of myelitis, we must not forget certain other etiological factors which formerly played a prominent part, but are now relegated to the background. This is true, for example, of colds. If we have to deal, as in certain of these cases, with marked cooling of the entire cutaneous surface, for example after sleeping in the open air during cold weather, the question of an intoxication due to sudden suppression of the entire cutaneous activity may come under consideration. It is more difficult to explain the positively observed cases of disseminated myelitis and multiple sclerosis following traumatism. In such cases our first thought would be that the trauma had mobilized some virus. The frequency of so-called myelitis after expeditions in war may be explained not alone by the hardships, but by colds, infectious diseases, injuries, abuse of alcohol, and syphilis. True myelitis has probably never occurred after a single sexual excess nor from suppression of the menses.

Pathological Anatomy.

With the section on pathological anatomy we come to the most difficult part of this, on the whole, so difficult subject. This is owing, in part, to purely extrinsic causes. In the cases of true inflammation of the cord (in which the etiological diagnosis is also well founded) death occurs very rarely in the acute stage, in which the recognition of the inflammatory nature of the disease would be most easy. Death usually occurs, if at all, at a period in which the inflammation has undergone resolution or cicatricial development has set in. Hence the pathological anatomy of acute myelitis is based either on the cases of rapidly fatal traumatic so-called myelitis, which are now recognized as non-inflammatory, or on cases of paraplegia which develop acutely, especially after syphilis, and which depend in the majority of cases not upon inflammation but upon thrombotic softening. For these reasons it is very easily understood that the histological descriptions of acute myelitis on the one hand, and of traumatic or vascular softening on the other hand, can hardly be distinguished from one another. The matter becomes still more obscure in chronic myelitis which has developed out of an acute attack or has been chronic from the beginning. Here we find only the results of retrogressive metamorphosis, especially the formation of cicatrices, and

histological examination has been unable hitherto to distinguish an inflammatory cicatrix from one due to traumatic destruction. We have already stated that the pathological histology of the cord in general is still involved in obscurity. This is due in part to the fact that, in softening of the cord, the preparation of sufficiently thin microscopical sections is attended with difficulty.

Hitherto very few cases have been reported in which the inflammatory nature of the disease of the spinal cord has been proven by the anatomicopathological findings, particularly by the demonstration of inflammation-producers. Clinically, almost all such cases were examples of a rapidly fatal Landry's paralysis. The findings in one case of Oettinger and Marinesco which was examined with all the latest technical auxiliaries, may be mentioned here. Clinically the case was one of Landry's ascending paralysis. The peripheral nervous system was anatomically normal. In the spinal cord the changes affected mainly the blood-vessels; throughout the entire length and breadth of the cord enormous numbers of leucocytes and other cells were embedded between the different parts of the vascular walls (perivasculitis). These cells often contained streptococci. The latter were also found in large numbers free in the tissues and in the cells which infiltrated the tissue of the cord. Smaller vessels contained thrombi and exhibited endarteritic processes; sometimes evidences of recent hemorrhage were found in the vicinity. Some of the ganglion cells also contained cocci. Otherwise they exhibited all the stages of cloudy swelling; the axis cylinder and protoplasmic processes were often torn off close to the cell, and there was usually also a change in the arrangement of the chromatophilic substance. The microbes were especially numerous in the central canal, in which the process probably extended very rapidly from below upwards; the disease was most pronounced in the lumbar cord.

This case, in which, as in the others mentioned above, there can be no doubt of the inflammatory nature of the disease, may be regarded as a model of the method to be pursued in the further study of acute myelitis. As a matter of course, such clear findings can be expected only in cases in which the disease of the cord is due, if not to the germs of the primary disease, at least to secondary living inflammation-producers. If we have to deal with an effect of a toxin of the primary disease, the matter cannot be decided offhand.

We may also, with tolerable certainty, infer a true inflammation when, as has been proven particularly in the examination of fresh cases of anterior poliomyelitis, the process extends only to the vicin-

ity of special vascular tracts of the spinal cord. The findings in recent disseminated encephalomyelitis may also be utilized in the same way.

These cases of acute inflammation of the spinal cord, which are histologically placed beyond doubt, form a very small, although extremely important, minority. If we do not wish to abandon completely a systematic description of the pathological anatomy, we must still adhere to the pictures exhibited by traumatic and vascular softening of the cord. In this direction the picture of myelitis, in all its details, has already been furnished by Leyden, Erb, and Pick, although with great reservation by the two latter authors. Under the same reservations, we may give the following description of the pathological anatomy of myelitis and which in its main feature is modelled after that of Erb and Pick.

We shall consider for the present only the so-called acute transverse, more or less diffuse myelitis. The adjective "transverse" indicates that the disease extends, at a certain level, over a large part of the transverse section without any regard to the systems of fibres. It never destroys the transverse section entirely but always leaves islets of intact fibres. The adjective "diffuse" refers to the extension of the inflammation along the length of the cord, and this may involve one or many segments. The extension along the transverse section may also vary greatly at different levels.

According to Erb we may distinguish in myelitis, from an anatomicopathological standpoint, three stages macroscopically: (1) Red softening; (2) yellow or white softening; (3) the stage of resolution, retrogressive metamorphosis, or formation of cicatrices. In the first stage, the cord is swollen as a whole at the site of inflammation; if a transverse section is made, it usually projects considerably above the level of the cut. The picture presented by the transverse section is generally indistinct, in many cases nothing can be recognized because the boundaries between the white and gray matter are obliterated. In not very severe cases the transverse section exhibits a speckled, marbled appearance, in which patches of extreme softening alternate with others which are more or less normal. If the inflammation affects the entire tranvserse section, the latter exhibits a homogeneous bright-red, reddish-brown, or chocolate color, according to the degree of decomposition of the extravasated coloring matter of the blood. In such cases we speak of a hemorrhagic myelitis. The softening may vary greatly in intensity in this stage. In the most extreme cases the nervous tissue simply flows out when the section is made; in the mildest cases we find merely œdema or increased juiciness of the cord. There are numerous transitions

between these extremes. In many cases the pia mater takes part in the inflammation.

In the second stage, that of yellow softening, an increase in the dimensions of the cord is no longer noticeable, and indeed it is often somewhat smaller. The red color has faded gradually into yellowish-red, yellow, or finally white. This change depends, on the one hand, upon the gradual absorption of the red globules and their coloring matter, on the other hand, upon the abundant development of so-called granular corpuscles. In this stage the softening of the cord has usually attained its acme, and the mass of detritus is made still more fluid by the inflammatory œdema which, according to Erb, is always present.

In the third stage the absorption of the degenerated medullary masses becomes more and more distinct, and the granular cells, which serve to remove the detritus, are not present in such large numbers as before. Now the dimensions of the cord necessarily diminish. Finally, if the longitudinal dimensions of the inflammation have been slight, this spot exhibits a narrow fissure; if a larger number of segments have been affected, the part in question may be seen passing from the upper or lower portions of the cord as a more or less flat band. With the destruction of the nervous substance proper, the supporting substance becomes more and more prominent, consisting as it does of blood-vessels, the neuroglia, and connective-tissue fibres. The transverse section thus assumes a grayer color, especially because these connective-tissue parts undergo notable proliferation and finally produce what is known as a spinal-cord cicatrix. At the site of this cicatrix the consistence is naturally increased, and to a varying extent according to the duration of the disease. The well-known secondary degenerations pass up and down from the locality of the cicatrix. It often happens that the morbid process does not terminate with the acute stage, and in such cases it spreads slowly at the borders of the primarily affected part. This constitutes the transition from an acute to a chronic myelitis.

In rare cases a cicatrix does not form at the site of inflammation, but a thin-walled cyst, filled with clear serum, is produced. Much more frequently a number of small cavities are found in the cicatricial tissue. A transition into an abscess—plausible as this would seem in a purely inflammatory disease of the cord—has not been observed hitherto in myelitis, with the exception perhaps of a few cases of gonorrhœal myelitis. Experimentally, as a matter of course, abscesses have often been produced.

After hardening in chromic acid the parts which exhibit retrogressive metamorphosis, cicatricial development, and secondary de-

generation become much more distinctly visible than when seen in the fresh condition. The acid stains the normal parts of the white substance dark brown to greenish-brown; the diseased parts remain light yellow. After this manipulation the extension of the process may often be ascertained very accurately. But it must be remembered that the chromic acid often allows the degeneration to appear much more pronounced than it is shown to be on microscopical examination. After hardening in chromic acid, if this is still possible, places in a condition of red or white softening exhibit a diffuse yellow color and, upon transverse section, a crumbly appearance as a sign of the intensity of the degeneration. A smooth cut can no longer be made.

As a matter of course these macroscopic findings will only be obtained if the disease is quite pronounced both in intensity and extension. If the inflammation is just beginning or if it affects very small, circumscribed parts of the transverse section, the macroscopic findings may be entirely negative. Here reliance must be placed on the microscope, and this is also true of simple post-mortem softening of the cord which was often regarded formerly as inflammatory in character. We have already remarked that the microscopical examination is attended with great difficulty for technical reasons as well as because of our slight knowledge of the pathological histology of the cord. The microscopical examination may be made upon the fresh cord, or after hardening, embedding, and staining of the sections. Granular corpuscles, amyloid bodies, and certain forms of medullary degeneration are recognized best in the fresh condition. In the hardened preparations we note the extent of the process transversely, the proliferation and disease of the walls of the blood-vessels, the amount of connective tissue and neuroglia, the swelling of the axis cylinders, and the secondary degenerations.

In the first stage, that of red softening, we find fulness and even distention of all the vessels, including the smallest ones; an escape of cellular elements at first into the walls of the blood-vessels, later into the surrounding tissues; numerous perivascular hemorrhages; extreme infiltration of the entire cord with red blood globules, and in part with white globules. The nerve fibres present all stages of degeneration. At first there is swelling and splitting up of the medullary sheath, so that longitudinal sections of a nerve fibre look like a rosary. The medulla stains less and less with osmic acid, finally not at all, and in the end it degenerates completely while more and more granules are making their appearance. In this stage the axis cylinders are often very much swollen, so that on transverse section a large round patch, the axis cylinder, is surrounded by a very nar-

row medullary rim, and finally the latter may be entirely destroyed. In transverse sections with Weigert's stain, the axis cylinders often look like a pale blot on blotting paper. The swelling of the axis cylinder varies in its different segments, so that it also looks like a rosary on longitudinal section. The neuroglia and the connective tissue which enters the cord with the blood-vessels show only the first stages of swelling, and the interstices between the individual nerve fibres are somewhat enlarged. The ganglion cells show cloudy swelling and granulation, their contours become coarser, the protoplasmic and axis-cylinder processes break off. The nucleus and nucleolus remain intact longest. Often the cells are with difficulty recognized as such. If a microscopic section can be made in this stage, it is seen that the degeneration is usually most marked at the centre, where all the parts are often soft and have fallen out of the section. The more we approach the periphery the more normal is the condition of the parts. Sometimes a narrow rim beneath the pia is found to be entirely normal.

The second stage, that of yellow softening, shows the medullary degeneration at its height. If the softening has progressed to liquefaction, only an imperfect picture of the transverse section can be secured, even after hardening and embedding the preparation. Where pieces of the cord are retained in place by the celloidin, the picture is a varied one. At one spot we see transverse sections of numerous, closely aggregated blood-vessels with thickened walls, and an abundant infiltration of small cells in the immediate vicinity. In other places are found pieces of the more or less markedly affected nervous tissue, bits of the medullary sheath, granular crumbs of pigment, swollen axis cylinders, intermingled with fatty detritus, red blood globules, and the products of their degeneration. Here and there is seen a normal nerve fibre. Finally, we find islets which consist entirely of proliferated neuroglia or connective tissue; in Weigert's preparations they assume a uniform yellow color. In the diseased tissue the ganglion cells appear as small homogeneous clumps in which the nuclei are often recognized with difficulty. As a matter of course the appearances vary with the greater or less intensity of the softening. In the slighter grades the markings of the transverse section of the cord are easily distinguishable and it is only in the centre that the greatest destruction is found. Sometimes the section is kept intact only by the framework of the blood-vessels and the neuroglia. In this stage examination in the fresh condition shows a very large number of granular corpuscles, also irregularly shaped drops of myelin, fat, blood pigment, and red and white blood globules.

The third stage, that of cicatrization, shows merely the remains

of the medullary degeneration, a great part of which has been absorbed. The foreground is here occupied by the proliferation of the neuroglia and the connective tissue proper, which gives rise to the cicatricial formation and finally occupies the entire transverse section of the cord. Corresponding to its origin the cicatrix always shows masses of thick-walled vessels and also remains of the medullary tissue, very often pigment clumps, more rarely a few well-preserved nerve fibres, and perhaps a few ganglion cells which can be recognized with difficulty. The cicatricial tissue also contains the so-called spider cells. The above-mentioned large cysts correspond exactly to those which develop in cerebral hemorrhages or vascular softenings. The smaller ones have a different origin. They are due to circumscribed necrosis of individual nerve bundles which sometimes attain large dimensions along the longitudinal axis. If the necrotic parts are absorbed, long cylindrical cavities develop. At certain periods of its development the necrotic part lies loose in the surrounding cord, like an osseous sequestrum. The amyloid bodies are found most frequently in this stage.

A few words will suffice for so-called *chronic* myelitis. As a primary disease—apart from the cases in which we really have to deal with a multiple sclerosis—it is very rare. Diffuse cases have been described in which there was thinning of the entire cord, with a very considerable increase of consistence. Under the microscope these cases also showed cicatricial-tissue proliferation of the blood-vessels, and the production of spider cells. In the majority of cases chronic myelitis follows an acute attack. Red and yellow softening are then found in those parts in which the disease of the cord is advancing, and cicatricial formation in the parts affected at an earlier period.

There are rare cases of clinically assured acute transverse myelitis which undergo complete recovery after the most severe symptoms have lasted for some time. In these cases the inflammatory origin is absolutely certain, because they almost always develop after an acute infectious disease. It must be assumed that the virus here gives rise merely to an inhibition of function, not to destruction of the parts of the cord, inasmuch as after actual destruction the human spinal cord is not capable of restoration.

After this detailed description of the pathological anatomy of transverse myelitis, we must again emphatically assert that all those lesions mentioned by writers as characteristic of myelitis possess no specific value, that the entire pathological anatomy of myelitis is based upon a very weak foundation, and that the above description (which follows Erb and Pick) possesses merely a didactic but no scientific value. To prove these statements we need merely ad-

duce the pathological findings in undoubted vascular affections and in acute compressions by trauma or tumor. It is very characteristic that the stages of red, yellow, and white softening which are said to correspond, in the cord, to various periods of an inflammation, have been long known in the brain as the results of embolism or vascular softening, and that the two conditions cannot be distinguished macroscopically from one another. It is also well known that the large serous cysts, mentioned above, are also found frequently in the brain after hemorrhages. Moreover, the microscopical appearances of those cases of so-called acute myelitis in which a vascular origin is undoubted agree entirely with those which have been ascribed to true myelitis. For this reason Tietzen goes so far as to assume a thrombotic origin in all cases of so-called acute myelitis. This also holds good of the cases of most acute softening after trauma or, as sometimes happens, in tumors. In such cases we find the abundant proliferation and disease of the walls of the vessels, the perivascular infiltrations and hemorrhages, the granular cells, and the proliferation of the neuroglia, which have been so often regarded as positive signs of a true inflammation. The identity of all these anatomical findings becomes still clearer when we pass from the stage of inflammation to that of cicatricial formation. It is impossible to distinguish a cicatrix of the spinal cord which is due to injury from one which has followed an inflammation. In brief, the anatomical picture regarded as characteristic of myelitis does not permit at any stage (apart from the very rare cases in which pus cocci are found) the diagnosis of a myelitis as opposed to a thrombotic or traumatic softening of the cord. The points which will decide us in favor of myelitis are especially the etiological factors and also certain peculiarities in the course of the disease. A decisive factor against myelitis will be the discovery (often unrecognized during life) of compression by a tumor or caries.

We are indebted to Küstner and Brosin for a careful anatomical examination of a fresh case of so-called *disseminated encephalomyelitis*. Gonorrhœa was the etiological factor. A large number of extremely small foci of degeneration were found, always very close to the blood-vessels. At first there was emigration of white blood globules and infiltration of the tissue, then circumscribed destruction of the nerve tissues, whose products of degeneration were mingled with the round cells. Then followed absorption so that at first only the neuroglia network was left over in the affected parts. At a later period circumscribed cicatricial development set in. Corresponding to the anatomical processes recovery occurs relatively often in these cases. We have already stated that the close relations of the spinal

affection to changes in the vessels and its etiology demonstrate the true inflammatory character of the disease in these cases.

Negative findings were formerly regarded as characteristic of the central form of Landry's paralysis, but the case above quoted of Marinesco shows what may be expected here from the employment of the most recent technique.

Symptoms, Course, and Termination.

Acute Transverse Myelitis.

It is impossible to give an accurate systematic history of acute transverse myelitis which will hold good for the majority of cases. This is owing to the fact that the morbid process is not confined to definite systems but may exhibit the most varied localization. Thus the disease may practically destroy the transverse section at a certain level, while in other cases it attacks only a part at varying levels. The longitudinal extension may sometimes be confined to one or two segments, in other cases it will involve almost the entire cord, the extent horizontally varying greatly at different levels. Furthermore, the disease may not be confined to one focus. Indeed, recent experience teaches that usually, in addition to one large focus, there are a number of smaller ones, even apart from disseminated encephalomyelitis proper. The smaller foci are situated not infrequently in the brain, particularly, according to Hoffmann, in the optic nerve. As the special symptomatology of spinal affections depends mainly on the location and on the transverse and longitudinal dimensions of the focus of disease, we can readily understand the variability of the clinical history of myelitis in individual cases. It would be entirely out of the question to give a detailed description of every possible case. In this regard an analysis of the individual case must be made by applying our anatomical and physiological knowledge of the functions of the different segments of the cord. In the following remarks we will first make a general survey of the symptoms occurring in every case of acute myelitis, and then will make a few definite statements concerning the symptoms at various levels. Finally we will give the symptomatology of disseminated encephalomyelitis and Landry's paralysis.

The disturbances which occur in acute inflammation of the spinal cord may be divided into sensory, motor, and trophic, and the first two groups may be subdivided according as the symptoms are those of irritation or of paralysis. The intensity period of development and the grouping of all these symptoms depend not alone upon the extent of the disease in the transverse and longitudinal directions,

but also very materially upon the rapidity of the onset of the disease. For example, in the cases of the most acute myelitis, also known as hemorrhagic myelitis (its inflammatory character is very doubtful), pain may be entirely wanting, while in the more subacute or chronic cases it is almost always present, although in a mild form and particularly as an initial symptom. In these cases the pains are usually confined to certain parts of the skin, but sometimes they are located in the back or exhibit an exquisite girdle character; they are tearing, lancinating, or dull and boring in character, often burning. In the latter event there is usually considerable hyperæsthesia of the corresponding part of the skin. If the disease progresses and distinct motor and sensory paralytic symptoms set in, the pains generally disappear. Intense and long protracted pains always arouse the suspicion of implication of the meninges and of the extramedullary posterior roots. We also know, however, that a disease of the second and third sensory neurons in the cord and brain may give rise to excentric pains, and that these may even appear in entirely anæsthetic districts.

Paræsthesiæ are more frequent than distinct pain in myelitis, and may appear at any time or be present during the entire course of the disease. They include the sensations of numbness, formication, and tingling in the limbs, sometimes the sensation as if a warm current of water were flowing from the proximal extremity of the limbs down into the fingers and toes. Complaints are often made that a certain part of the limb is swollen, for example, the foot. This sensation is sometimes so distinct that the patient can be convinced with difficulty of its subjectivity.

In point of time anæsthesia always follows the irritative symptoms, if the latter are present. It is often very indistinct in myelitis and may even be absent during the entire course of the disease. In other cases it extends rapidly over the entire region which is innervated from the level of the morbid process. As a matter of course, this will happen only if the entire transverse section is affected. It is very important, however, to know that particularly at the upper extremity of the morbid process the transverse section is usually affected only partially, and that the limits of cutaneous anæsthesia will then lie considerably below the domain of the uppermost diseased segment of the cord. Hence we are hardly justified in drawing an inference, from the extent of the anæsthesia, concerning the upper limits of the disease in the cord. If the anæsthesia spreads slowly in the special case, it usually extends from the end of the limb towards the trunk.

The anæsthesia of myelitis is usually bilateral but does not always

extend to the same height on both sides. It is only in very rare cases that the myelitic process is confined to one-half of the cord. In these cases, as in Brown-Séquard's paralysis, the pains and paræsthesiæ may be found, as root symptoms, on the same side as the disease, and the anæsthesia upon the opposite side. Not so very rarely the anæsthesia does not affect uniformly all qualities of sensation. As in syringomyelia and certain peripheral diseases, it may be confined to the sense of pain and temperature, while tactile sensation remains unaffected. In such cases, for example, there may be total anæsthesia of the leg and of the posterior surface of the thigh, and partial anæsthesia of the anterior and inner surfaces of the thigh. In mild cases, the point of contact may merely be localized falsely or the sensation of pain may be delayed. Beyond the limits of total or partial anæsthesia there is sometimes found a narrow zone of distinct hyperæsthesia, which is arranged like a girdle on the trunk. This is very important in local diagnosis, because it indicates the level of the disease in the cord. But it is much less distinct in myelitis than in diseases which, like meningeal tumors, for example, lead primarily to a lesion of the roots of the nerves.

Among the motor symptoms the irritative phenomena likewise precede the paralytic symptoms. Here we have to deal first with very painful but rapidly subsiding cramps, which are found mainly in the muscles of the lower limbs (particularly the calves) and resemble closely the cramps produced by exhaustion or observed in alcoholic cases. It is also possible that these cramps are produced by a primary irritation of sensory tracts, particularly of the posterior roots. The cramps may develop into protracted tonic spasms of the entire limb, but this always indicates an associated lesion of the anterior roots. Involuntary twitching movements are very frequent in the completely paralyzed and perhaps completely anæsthetic lower limbs. These twitchings consist very often of a flexion of the thigh, which occurs rapidly in one case, and in another may be peculiarly slow. If the paralysis and anæsthesia of the legs are not complete, the twitchings may be attended with pain; even when both are complete, the movement may be brought to the consciousness of the patient by the concussion conveyed to the entire body. Tetanic rigidity of the entire body, described by Pick, can occur only in very diffuse myelitis and, in my opinion, should arouse the suspicion of a notable implication of the meninges.

The motor irritative symptoms are followed by paralysis. Paraplegia is typical of myelitis, particularly of the lower limbs, rarely of all four extremities. As a matter of course the extent of the paralysis depends upon the height of the morbid process and, if the latter

extends approximately through the cord in a transverse direction, all the muscles will be paralyzed which are innervated by the diseased portion of the cord and the parts below it. The following remarks are merely general in their application, and a special study of the grouping of the paralyses at various levels of the disease will be made later. The paralysis develops rapidly in foudroyant cases and may become complete in a very short time; not infrequently it appears during the night, without having been noticed by the sleeping patient. More frequently an apparently slowly developing paresis is suddenly converted into complete paraplegia. At first it may be perfectly flaccid and the well-known contracture appears only after a certain lapse of time, at least in dorsal and cervical myelitis and apart from certain special conditions which will be considered later. The contracture finally leads to rigid spastic tension, usually in extension, more rarely in flexion of the legs. The flexion contracture is usually attended with rigid adduction contracture of the thigh. Both interfere materially with our efforts to keep the patient clean, and hence this contracture is very unfavorable from a prognostic standpoint. In subacute cases the paralysis sets in more gradually, and the contracture may keep pace with the former. It may also happen that one leg is paralyzed after the other, and, like the anæsthesia, the paralysis may extend gradually from the toes to the ankle, so that the foot is paralyzed while the thigh can still be moved. In the rare unilateral myelitis the paralysis of the muscles is found, as a matter of course, upon the side of the lesion opposite to the anæsthesia.

The relation of the sensory disorders to the paralysis is very variable, especially from a topographical standpoint. Motion is almost always affected much more than sensation. Anæsthesia may be entirely absent even in severe paralysis, or, if it was present at the outset, it may disappear rapidly while the paralysis persists. If the morbid process slowly advances, the anæsthesia often lags behind the paralysis. For example, the legs may be completely paralyzed while sensation is still intact upon the anterior surface of the thighs. Even at the height of the disease the motor and sensory disturbances may not correspond in distribution. All these phenomena depend, of course, upon the irregular extension of the disease over the transverse section of the cord.

Irritative and paralytic symptoms, whether motor or sensory, are not separated so sharply from one another as is here represented. If the disease develops by fits and starts, the paralytic symptoms will be constantly accompanied by new irritative symptoms. The muscles which depend directly upon the inflamed parts of the cord undergo degenerative atrophy if the inflammation has lasted for some

ness), that we are compelled to take into consideration a possible failure of trophic influences on the part of the cord.

If the myelitis is situated in the cervical cord, paralysis of the phrenic nerve may cause death quickly from asphyxia.

Myosis and narrowing of the palpebral fissure are observed when the lesion involves the first dorsal and adjacent segments. The occurrence of optic neuritis has been noted frequently in myelitis and has been interpreted in various ways. It is certain that it does not occur in a transverse myelitis which is confined to the cord. We always have to deal with a coincident disease of the optic nerve and the spinal cord, *i.e.*, with a disseminated inflammation or at least one which appears in several foci.

The general condition may suffer very considerably in acute transverse myelitis. Foudroyant cases, which have a tendency to spread diffusely, are often accompanied by fever. Not so very rarely, and particularly in Landry's paralysis, the spinal symptoms proper are preceded by a prolonged stage of general disturbances, such as malaise, anorexia, and slight fever. If decubitus and cystitis develop, fever will always be present, sleep will be disturbed, and the nutrition will be impaired. But if the inflammatory focus is small and its location is relatively favorable, and if the development of these serious complications is avoided, then the general condition will usually be good after the acute stage has subsided.

The course of the disease may vary greatly, as is evident from what has been said in the section on the pathological anatomy. This is true of the very onset of the affection. In a series of cases paraplegia—the most important and typical symptom—develops acutely, not infrequently at night, so that it is unnoticed by the patient, who does not become aware of his condition until he awakens in the morning. In these cases, unless there are good reasons to the contrary, we should suspect a vascular softening rather than a true myelitis. In other cases the disease does not reach its height at once, but several apoplectiform attacks occur and their effects are added to one another to complete the clinical history. In other cases from a few to many hours elapse before the paralysis is complete. At first the patient merely feels a heaviness or tired feeling in the legs. Indeed, this condition may disappear temporarily after resting, but it soon returns. Then he is unable to stand erect, must lie down, and the paralysis becomes complete. In cases which run a still slower course and which should be called subacute rather than acute, the paralysis begins in one leg, ascends slowly in this limb, and then extends to the other limb. In such instances two to three weeks may elapse before the paralysis is completely developed. There may also

be remissions which border on recovery, but these are always followed by exacerbations. If, as rarely happens, there is no complete paraplegia but merely paresis of the lower limbs, it may still be possible to move the toes while the thighs are entirely helpless, although the paralysis of the legs almost always passes, as we have said, from below upwards. This is probably owing to the fact that much less power is necessary to move the toes than to move the thigh.

In the more slowly developing cases there may be considerable pain and various paræsthesiæ, especially before the paralysis. The remaining symptoms, the anæsthesia, paralysis of the bladder and rectum, and trophic disturbances, follow the initial symptoms in an equally variable manner.

The termination of acute transverse myelitis is as variable as are the onset and course. It results quite often in death. This may occur in a few days in the most acute cases, which spread rapidly, are often attended with high fever, and in which so-called acute decubitus sets in. These are the cases which were described by earlier writers as acute central myelitis (myelitis generalis seu diffusa) and which exhibit many points of resemblance to Landry's ascending paralysis. In other cases the process does not spread so rapidly but it progresses uninterruptedly, finally reaches important centres, particularly that of the respiratory muscles, and the patient dies of asphyxia. In a third series of cases—the most numerous among the fatal ones—death results finally from the complications, such as cystitis and pyelonephritis, decubitus, or from the general impairment of nutrition (marasmus). Lumbar myelitis is more unfavorable in this regard than are other forms, because in it the bladder disturbances are usually more pronounced.

In a large number of cases of acute myelitis a fatal termination does not result. As a matter of course, the progress and termination of the disease may vary greatly after the end of the acute stage. The disease may continue permanently at its acme, *i.e.*, a condition of complete paralysis. This occurs most frequently in dorsal myelitis. We then find paralysis of the legs, with contracture in extension, more rarely in flexion, and greatly exaggerated reflexes. In lumbar myelitis flaccid paralysis, muscular atrophy, and bladder disturbances remain. But this flabby paraplegia is rare as a permanent sequela of myelitis because the patients usually die. This is also true of the occurrence of permanent paralysis of all four limbs in cervical myelitis. On the whole a persistence of the disease at its acme is rare in those cases in which life is preserved. In the writer's opinion this should arouse the suspicion of a compressing factor, perhaps a slowly growing tumor of the meninges. After the acute stage has passed, at

least in the large majority of cases of dorsal myelitis, a considerable improvement soon occurs. The patients again learn to stand and walk, at first with crutches, then without them. But the legs always remain weak and especially stiff, and are the seat of very distinct or more latent contractures. The tendon reflexes exhibit a clonic character, and the walk is either spastic-paretic, so that the feet are dragged and scrape the floor, or it is purely spastic, at every step the heel being lifted at once from the floor so that the gait becomes somewhat dancing in character. These are the cases of so-called spastic spinal paralysis, which are due, in the majority of cases, to multiple sclerosis or to a dorsal myelitis which has run its course. In these cases of myelitis we almost always find disorders of the vesical and rectal functions and, on careful examination, disturbances of sensation. In the most favorable event the weakness and stiffness may disappear almost entirely and then the increased tendon reflexes alone remain. This is especially frequent in paraplegia due to spinal caries, which is only apparently a myelitis. The termination in complete recovery is extremely rare in acute transverse myelitis, but it has been observed in cases which were clinically so diagnosed. The expert who knows the remarkably slight tendency of the cord to recovery even in very mild affections will be inclined to believe in an error in diagnosis in such cases. Usually they are due to a multiple neuritis or to caries of the spine. But, as we have already remarked, there are undoubtedly cases in which we must finally accept the diagnosis of transverse myelitis, and in which recovery occurs. Pick and Oppenheim have pointed out that the favorable cases usually have a distinct infectious origin. We have observed a case of transverse myelitis after measles which terminated in perfect recovery.

This completes the description of the clinical history of myelitis in general, and we have already considered those varieties which are characterized by the greater or less rapidity and malignity of their course, by the more or less complete extension of the disease over the transverse section. It remains to give a brief analysis of those principal varieties of transverse myelitis which depend upon the location of the disease along the longitudinal section of the cord. We distinguish dorsal, lumbar, and cervical forms of myelitis. Only the general types can here be given. In regard to the variations which depend upon the exact site of the disease at definite levels, we refer to the remarks made concerning tumors and injuries of the spinal cord. The following refers only to those cases in which the disease has involved at least the entire transverse section of one segment, in other words, to that stage of the disease in which it has reached its height.

Dorsal Transverse Myelitis.—This is the most frequent variety of myelitis, probably because the dorsal cord forms the largest division of the entire organ. In almost complete transverse lesion—the most frequent event—there is complete paralysis below the site of the disease at first flaccid, then growing gradually spastic. The contracture is usually in extension, rarely a flexion contracture of the legs and thighs is combined with adduction contracture of the thighs. The latter cases have a much more unfavorable prognosis. The paralysis of the limbs remains permanently flaccid in rare cases only (complete transverse lesion). At the height of the disease flaccid paralysis and degeneration reaction would necessarily develop in the muscles, but this escapes observation in the intercostal muscles. In disease of the lower dorsal cord, electrical disturbances may be demonstrated in the abdominal muscles. At the height of the disease sensation is abolished up to the level of the corresponding segment of the cord, although the sensory disturbance in many cases does not extend quite so high as the paralysis. A distinctly circumscribed zone of hyperæsthesia is rarely found in myelitis. There may be girdle pains or girdle paræsthesiæ. In very acute myelitis the tendon reflexes may be abolished during the first week (shock), but they are soon restored, then increase and acquire the intensity to which Brown-Séquard applied the term spinal epilepsy. It is also certain that they may be permanently abolished in total myelitic softening of the dorsal cord. At first there is usually retention of urine and catheterization is necessary. Later incontinence of urine, ischuria paradoxa, or involuntary evacuation of the bladder in gushes may set in. If the disease pursues a favorable course, mere weakness of the detrusor or increased and imperative desire is alone left over. The bowels are very constipated and usually must be evacuated artificially. The occurrence of bedsores is due mainly to the more or less distinct anæsthesia and secondarily to the intensity of the bladder symptoms, but with good nursing it may usually be prevented.

The disease may terminate fatally in the acute stage or from marasmus; life may be preserved, all the symptoms persisting (rare); or a spastic paralysis remains. Complete recovery is rare.

Lumbar Myelitis.—If the myelitis is total and extends over the entire lumbosacral tract, there is paralysis of all the muscles of both legs. The paralysis always remains flaccid, and is combined with muscular atrophy and degeneration reaction. The anæsthesia extends posteriorly to the crest of the ilium, anteriorly to a little beyond the inguinal fold. The location of the pains corresponds to the upper borders of the anæsthesia, but they may also radiate into the legs. The tendon reflexes are permanently abolished. The bladder exhibits

complete incontinence, and the functions of the rectum are performed as in severe dorsal myelitis. On account of the severe bladder disturbances decubitus is much more frequent than in dorsal myelitis; it is equally difficult to heal after it has once developed. Hence death in lumbar myelitis occurs more frequently and at an earlier period than in dorsal myelitis. If life is preserved the flaccid paralysis, vesical paralysis, and impotence persist. The ability to walk is never restored. If the myelitis is confined exclusively to the lumbar cord, flaccid paralysis with degenerative atrophy occurs only in the muscles of the thighs; the paralysis of the legs is not attended with atrophy and may be spastic in character. The sensory disturbance has the same distribution as in total lumbosacral myelitis. The patellar reflexes are absent, but there may be ankle clonus. The bladder and rectum show the same symptoms as in dorsal myelitis.

In pure *sacral* myelitis the muscles of the legs, the flexor cruris, the glutei, and the peroneal muscles show flabby paralysis and degeneration reaction; the other nuclei of the lower limbs are unaffected. The anæsthesia is confined essentially to the posterior surfaces of the legs. The Achilles tendon reflex is absent, the patellar reflex may be intact. There is complete paralysis of the bladder and rectum.

On account of the shortness of the lumbar enlargement these varieties—lumbar and sacral myelitis—are very rare in a pure form. It is hardly possible that a myelitis could be confined to the conus terminalis. It would be manifested by paralysis of the bladder and rectum, impotence, anæsthesia in the region of the anus, perineum, scrotum, penis, and the posterior surface of the thighs, perhaps by degenerative paralysis in the distribution of the sciatic nerves.

Cervical Myelitis.—If the entire cervical enlargement is involved, there will be flaccid paralysis of both arms with degenerative atrophy of the muscles and degeneration reaction. The triceps reflex is abolished. There is spastic paralysis of the lower limbs and trunk. Sensation is abolished on the trunk as far as the second rib anteriorly and the spine of the scapula posteriorly, the arms are completely anæsthetic; the palpebral fissures and pupils are narrowed. If the lower half of the cervical enlargement alone is involved, the movements of the shoulders and elbow-joints are unaffected, the forearms and hands are paralyzed. The anæsthesia affects the ulnar halves of the arms and hands; the pupils and palpebral fissures are narrowed. If the inflammation is located in the upper half of the cervical enlargement, the upper limbs are completely paralyzed and anæsthetic, but the muscles of the arm and shoulder alone are atrophic, while the hands and fingers are contractured. In every case of cervical myelitis

the respiration is very difficult, because all the intercostal muscles are paralyzed; priapism is usually present. The prognosis of cervical myelitis is much worse than that of dorsal myelitis.

Myelitis is most dangeous when it is located above the cervical enlargement. The phrenic nucleus is affected at the fourth cervical segment and asphyxia is then unavoidable, at least when the transverse lesion is approximately total. If myelitis of the upper cervical cord runs a slow course, the first symptoms may be degenerative atrophy of the sternocleidomastoid and trapezius muscles with difficulty of moving the head, while spastic symptoms are observed in the arms and legs. True myelitis is very rare in this region.

In a few cases of cervical myelitis, when there is a very peculiar localization of the disease in the transverse section, isolated paralysis of the arms has been observed without paralysis of the legs, so-called brachial paraplegia. As a general rule brachial paraplegia indicates a disease of the roots of the brachial plexus outside of the cord, and in these cases, as a matter of course, there is flaccid paralysis of the arms with degenerative atrophy. We cannot enter here into a description of all the possible varieties which may be due to partial disease of the transverse section. A few points have been mentioned above; the rest must be left to the physiological analysis of the individual case.

Chronic Myelitis.

The description of so-called chronic myelitis may be very brief. We agree entirely with Oppenheim, who believes that the majority of cases so called are really cases of multiple sclerosis, that a second and smaller series of cases are due to the chronic spread of acute myelitis, and that transverse myelitis, chronic from the beginning, is a very rare disease, if indeed it occurs at all. But as some of the most recent writers, for example Gowers, entertain a different opinion and still regard chronic myelitis as a frequent disease, we must defend our views somewhat in detail. They are based upon the historical development of the doctrine of this form of myelitis in the last few decennia. In 1878 Erb included tabes, multiple sclerosis, primary sclerosis of the lateral tracts, amyotrophic lateral sclerosis, and the combined system diseases in the category of chronic myelitis, and differentiated these diseases from the primary form of myelitis, in part for clinical and anatomicopathological reasons, in part for purely didactic reasons. This author, on account of the imperfect knowledge of the diseases at that time, also included among the varieties of chronic myelitis many cases of syringomyelia and of chronic peripheral neuritis. Pick made a much sharper differentiation of

these affections. He knew that Hallopeau's peripendymal sclerosis was really syringomyelia; he recognized chronic peripheral neuritis, and differentiated from myelitis spasmodic tabes of the aged (Demange) as an arteriosclerotic affection. A part of the cases described as chronic cervical myelitis he included in the category of hypertrophic cervical pachymeningitis. The diseases of the cord which result from compression (tumor, caries) are not divided sharply by him from inflammations proper. Pick reserves the term chronic myelitis (he also recognizes its frequent development out of acute myelitis) for chronic transverse myelitis, which may be confined to small foci or gradually involve large portions of the cord, and the so-called annular sclerosis (sclérose corticale annulaire of Vulpian). We will here discuss the latter affection.

In the majority of cases we have to deal with an inflammation which penetrates the cord from the membranes or the cortical vessels, *i.e.*, a so-called meningomyelitis. Rarely is there a primary inflammation of these parts in the sense of the pseudosystematic combined system diseases (Leyden and Goldscheider). It is also to be noted that the entire cortical region is occupied by ascending as well as descending degenerated fibres, so that if a small transverse focus in any part is overlooked, the remainder of the cord may suggest a primary annular sclerosis.

There remains only the chronic transverse (and usually very diffuse) myelitis. This is said to have a tendency to spread gradually over the entire cord and then to extend into the medulla oblongata. In by no means a few cases of multiple sclerosis, in which life is preserved for a long time, it is found that the foci coalesce to a greater or less degree. In this way the picture of a very diffuse sclerosis develops and in some sections this sclerosis is total. If at the same time foci are present in the medulla oblongata or the spinal foci gradually ascend to the medulla, the clinical history of multiple sclerosis will naturally be produced. If the foci are confined to the cord, as is not impossible, we find the clinical history of a chronic myelitis (apart from the acute exacerbations), and indeed this is also true from the anatomical standpoint. But there is no doubt that such cases are very rare.

After all these considerations there are left, in the category of chronic myelitis, only the chronic continuations of the acute form and perhaps some very rare cases which we must, for the present, still interpret as examples of primary chronic myelitis. As a matter of course these cases do not differ materially in their clinical symptoms from those of acute myelitis, for each individual symptom may occur in both forms. The slow course of the inflammation will give

rise, in the main, to three peculiarities of chronic myelitis. In the first place the individual stages described above will be divided more sharply from one another. In particular, the stage of sensory and motor irritative symptoms may continue alone for a long time before paralytic symptoms make their appearance. In the second place the inflammation is more apt to be confined for a considerable period to individual parts of the transverse section and to give rise to corresponding clinical symptoms, for example, to spinal hemiplegia or, as Gowers describes it, to Brown-Séquard's paralysis, or to the above-mentioned brachial paraplegia. In the third place chronic myelitis naturally begins with very slight symptoms which gradually increase, while in acute myelitis the disease reaches its height at once and then, if any change occurs, takes a turn for the better. As a general thing pareses will continue for a long time in chronic myelitis before real paralyses develop. Sensation is often unchanged, and there is more apt to be simple weakness of the bladder than paralysis. For these reasons it is usually possible to prevent bedsores.

The general condition is almost always unaffected until the last stage. At the end all the symptoms attain great severity and marasmus then sets in. The flexion contractures, which are the source of much annoyance in nursing, are more frequent in the final stage of chronic myelitis than in the acute form.

The duration of chronic myelitis may be very great; the disease often lasts ten years or more. Not infrequently there are remissions, either temporary or permanent, and the disease may then continue as so-called spastic spinal paralysis. True remissions probably occur only in multiple sclerosis. In typical cases death is due to implication of the cervical respiratory centres of the medulla oblongata, or to complications. Recovery is impossible.

Landry's Paralysis.

We refer the reader to the remarks already made concerning Landry's paralysis in the historical, etiological, and anatomicopathological sections of this article. Here we shall discuss only the central form of ascending paralysis, disregarding the neuritic varieties. This intention cannot be carried out strictly, however, because there are often combinations of central and peripheral disease, and their symptoms then enable us to recognize their mixed character.

We may repeat once more that in this paralysis the connection between an infection and the spinal disease is usually very distinct, and would recall Marinesco's extremely instructive post-mortem findings in a case of this kind. In 1859 Landry described the clinical course of typical cases, and his masterly description has been

changed but little by subsequent observers. It is very characteristic that the spinal symptoms proper are usually preceded by a prolonged, ill-defined prodromal stage, which lasted eight days in one of the writer's cases. There is general malaise, with slight fever, anorexia, and drawing pains in the limbs. The spinal symptoms begin in the lower limbs, particularly in the feet. Paresis appears first in the feet and gradually spreads upwards, followed by paralysis. The affection of the feet is followed by that of the legs and thighs, then of the trunk and arms; then notable disturbance of respiration and symptoms of asphyxia set in, and the drama closes with disturbance in deglutition, articulation, phonation, and the action of the heart. A short interval usually elapses between each successive invasion of the different sections of the body. The paralysis does not always occur simultaneously in both lower extremities nor does it spread uniformly along the limbs. The disease rarely extends farther up, although cases have been described in which the facial nerve and ocular muscles were paralyzed. All the muscles mentioned are not paralyzed in succession in every case; it is not so very rare to find that some muscles escape or are merely paretic. In other cases the disease progresses by jumps. For example, it passes from the feet directly to the thighs, trunk, and arms, leaving the legs intact. The paralysis is always flaccid. The tendon reflexes are never exaggerated, in the majority of cases they are absent. They are demonstrable in a few cases, probably in those which run a very rapid course.

In typical cases the electrical excitability of the paralyzed muscles is unchanged. This probably depends on the fact that, on account of the rapid course of the disease, there is no time for the occurrence of the degeneration reaction. Slight disturbances of electrical excitability have, however, been observed in slowly progressing cases and particularly in those which recovered; but distinct disturbances of electrical excitability always indicate that we have to deal with the peripheral neuritic form of ascending paralysis. Muscular atrophy may also occur in such cases. Oppenheim has observed on several occasions that, upon electrical stimulation of the peripheral nerves, muscular contraction was produced early but did not increase with increasing strength of the current—*i.e.*, the minimum and maximum of nerve irritability lay close to one another. Perhaps this was due to rapid exhaustion of the electrical irritability of the nerves.

The disturbance of sensibility is moderate but is often distinct. Paræsthesiæ and slight impairment of tactile sensibility are especially frequent at the extremities of the limbs. In one of the writer's cases violent pains were also present, particularly in the prodromal stage. In other respects the case was clinically pure, but the pains

probably indicated a combination with peripheral neuritic processes.

In the absence of pronounced sensory disturbances bedsores usually do not develop. Pronounced disturbances of the bladder and rectum are absent in Landry's paralysis, although slight disorders of micturition may be present.

The sensorium is clear until the end, unless some febrile complication or asphyxia leads to unconsciousness. The patients are often extremely anxious, excited, and wakeful, especially after respiratory disturbances have set in. The organs of special sense are not involved.

The disorders of deglutition are the most severe among the bulbar symptoms. Solid particles are swallowed better than fluids, which usually regurgitate through the nose. The patient is always in danger of suffocation during meals. The disturbance of articulation and phonation may finally become so pronounced that the patient cannot be understood. Vigorous coughing is impossible. Great interest attaches to the fact that enlargement of the spleen is very often present. In rare cases the disease pursues a descending course. It begins at the medulla, then descends to the arms, but usually does not reach the lower limbs on account of the early death of the patient. We have already spoken of its progress by fits and starts, and would merely remark in addition that, in cases which are evidently closely allied, the process may begin simultaneously in parts remote from one another—for example, the medulla oblongata, the cervical, and the sacral cord.

The disease may run its course from the first spinal symptoms to the fatal termination in a very short time (two to three days); in other cases a few weeks elapse. In a third series of cases the disease reaches a certain height (even severe bulbar symptoms may develop), then it comes to a standstill, and regresses to complete recovery. The favorable cases always arouse the suspicion that we have to deal with the neuritic form of Landry's paralysis. This is assured when, as frequently happens, we have observed pronounced disturbances of electrical excitability, violent pains, and tenderness of the nerves.

Death may result from asphyxia, rarely from cardiac paralysis. "Schluck" pneumonia often sets in or the primary infection *per se* leads to the fatal termination.

Acute Disseminated Encephalomyelitis.

This disease (Leyden's acute central ataxia) is a myelitis with numerous small foci in the cord, pons, and usually the cerebrum. Westphal has described cases in which the foci were confined to the cord. These, if the foci are so numerous as to produce distinct symp-

toms, can hardly be distinguished from cases of simple transverse myelitis. The characteristic feature of the clinical history is furnished by the combination of bulbar symptoms, perhaps also of cerebral symptoms, with those dependent on disease of the cord. These cases alone will be discussed here in detail. Although the first carefully observed cases were published twenty-five years ago, as is shown in the historical *résumé* given above, and subsequently very accurate clinical communications were furnished by Leyden concerning his so-called acute ataxia, a knowledge of the disease has not been diffused largely in the profession. This is only natural, because even the latest and most complete text-books on nervous diseases mention this affection cursorily or not at all. In fact, however, disseminated encephalomyelitis possesses great practical importance. In the first place it is, in our experience, more frequent than so-called transverse myelitis. This is particularly true if we speak of myelitis only in cases which, etiologically, are positively recognized as inflammatory (those which follow infectious diseases). Moreover, these cases usually exhibit from the outset an extremely serious appearance, while in many of them complete or almost complete recovery ensues. Hence the experienced practitioner is enabled at an early period to allay the fears of the family concerning the course of the disease. As cases of this disease present some variations in regard to etiology and symptomatology, we may best describe the main features of disease by recounting the clinical history of a few cases which have come under our own observation.

Case I.—Disseminated Encephalomyelitis after Varicella.—A girl, aged 4 years. Varicella ten days after a sister had been attacked. After the primary disease had been recovered from, the patient was apparently well, but somewhat dull. March 5th, 1892, on awaking in the morning she was in same condition as at the present time (March 8th). Sensorium clear; answers all questions correctly. Speech is typically scanning, nasal, and sometimes dysarthric; the beginning of a word is often explosive. Movements of face, tongue, and eyes are normal; there is no nystagmus; deglutition is good. In sitting, the head shows tremulous rotatory movements, and vigorous shaking when the patient fixes an object. Intention tremor is present on moving the arms, but it is mixed with widely excursive ataxic movements. When the patient is lying in bed there are vigorous ataxic movements of the legs on attempting to get the patellar reflex and static ataxia of the legs on rising from the bed. On attempting to hold the patient erect, there is at first violent shaking tremor of the legs, then of the trunk, arms and head, and unless supported she would fall at once. There are true ataxic movements of the legs on attempting to walk; sitting is also impossible. The patellar reflexes are normal. March 13th, decided improvement. Speech good, only slight tremor of the arms. Sitting again possible. March 15th, still typical intention

tremor of the arms; walking possible but with tottering and stamping. The child is mentally very excitable. March 20th, slight tremor of arms and hands; standing and walking good. Recovery soon became complete and permanent.

Case II.—Disseminated Encephalomyelitis after Poisoning with Gas of Unknown Character.—An employee of a gas company, aged 43 years, while making ammonium sulphate became unconscious and was found in that condition. The nature of poisonous gas could not be discovered. After admission to hospital he was unconscious for a few days, then slight consciousness, paralysis of the limbs. Discharged April, 1892, ostensibly cured. According to statement of the lodge physician, he was in the same condition as at present time (June 3d, 1892). Walk notably affected; after sitting for a long time the feet are first glued to the floor and the legs are dragged after him; gradually there is increasing tremor of the legs, which then extends to the trunk, arms, and head, so that the patient would fall if not supported. At the beginning of the tremor the heel is lifted from the floor so that only the tip of the foot is in contact with the floor (typical spastic gait). No Romberg's symptom, but there is the same tremor of the entire body in standing as in walking. Very vigorous ataxic movements on moving the legs in the recumbent position. Paresis of the legs, no contracture. Extreme clonus of the patellar and Achilles tendons; spinal epilepsy. Coarse intention tremor of the arms, sometimes slight ataxic symptoms; tremor in writing; paresis of the arms. Pronounced scanning speech, also tremor of the face in speaking. Tongue normal; no nystagmus. Special senses and intellect normal. Other findings normal. April, 1893, material improvement in every respect. Gait at first spastic, later marked tremor and tottering. The legs are not dragged until the close of a half-hour's examination, but are still paretic. Patellar and Achilles clonus, but not so severe as before. On movements of the legs in the recumbent posture there was no intention tremor as long as the legs were held quietly in the air. Arms almost well, but distinct intention tremor in the left arm. Speech still scanning and somewhat dysarthric. Slight tremor of the head. April, 1894, very striking improvement. Gait at first normal, slightly spastic after exhaustion. No tremor in walking or moving the legs in the recumbent position. No patellar clonus; slight clonus of Achilles tendon. Good power in the legs. Normal findings in the arms and head. Speech still scanning. Patient resumed his work, but grew tired very rapidly and soon abandoned it. At the end of April, 1894, all the symptoms were again very severe. Gait exquisitely spastic; spinal epilepsy. Strong tremor of left leg, not of right, in moving in recumbent position. Improvement continued in the arms, and soon returned in all other parts. In June, 1894, another relapse occurred after an attack of diarrhœa lasting several days.

This patient's wife suffered from multiple sclerosis, as shown by spastic paresis with tottering gait, intention tremor of the arms, scanning speech, and slight disturbance. The course was chronic progressive.

Case III.—Disseminated Encephalomyelitis after Scarlatina.—A girl, aged 7½ years. October, 1887, scarlatina without diphtheria; during

its course there occurred sudden unconsciousness (impossible to ascertain whether nephritis was present), which lasted thirty days. Great restlessness, delirium, tonic contractures of the arms and trunk. Then consciousness cleared up, but complete aphasia set in; the child expressed her desires by weeping. Pronounced, constant choreic movements, especially of right side; bladder and rectum intact. Legs at first completely paralyzed; arms could be raised somewhat with marked ataxic movements. Gradual increase of power, but persistence of ataxia. Christmas, 1887, sitting barely possible; pronounced shaking of the head. Speech is gradually learned again. February 13th, 1888, intelligence good. No nystagmus or other ocular symptom. Speech scanning and dysarthric. Slight cerebral paralysis of right facial nerve. Left arm stronger than the right, which exhibits distinct ataxia. Legs not paretic. Vigorous reflexes, no clonus. Most marked ataxia of the legs on moving them in the recumbent position; gait ataxic and tottering, with forcible stamping; walking possible only with assistance; standing possible with feet widely separated. Sensibility intact. April, 1888, improved. Could walk a few steps without assistance. In November, 1888, gait still stamping. Distinct ataxia in right arm; writing done poorly. Speech scanning, no longer dysarthric. April, 1890, gait somewhat uncertain, no distinct ataxia; nothing abnormal in hands and arms. Speech scanning. Findings approximately the same in April, 1896.

The writer (Bruns) has recently observed another case, similar to Case III., in a boy of two years after pertussis. Complete recovery occurred. Here the choreic stage was especially distinct.

From these briefly sketched cases it is possible to gain a good idea of the disease and its different varieties. From our own observations and those of others the following general statements may be made concerning the symptomatology of disseminated encephalomyelitis: The disease almost always begins during or immediately after an acute infectious disease or a toxæmia, and its frequent occurrence in childhood is explained by its close relations to the infectious diseases. It always begins acutely; in the first case above reported it began at night during sleep. In the majority of cases the symptoms of the first stage indicate a serious implication of the entire central nervous system; these were in Case II. prolonged unconsciousness and at first general paralysis; in Case III. unconsciousness with restlessness and delirium. Then the unconsciousness gradually disappears and sometimes, as in Case III., a stage appears which reminds us at first of a severe chorea. Total aphasia may be present at this time (Case III.), and optic neuritis has also been observed in several cases. In these severe cases it is only after the lapse of weeks that the disease reaches the stage which is most characteristic and from which the name acute ataxia has been derived. But this does not always hold good.

In a series of cases, for example in Case I., the severe cerebral symptoms may be absent and the disease then begins, if we may so express ourselves, with the second stage. According to Leyden, the clinical history then exhibits the following typical symptoms: (1) Ataxic movements of the legs or disturbances resembling intention tremor, while standing, walking, or moving the limbs in the recumbent position; similar symptoms in the arms on making grasping movements; severe tremor of the head. (2) Disorders of speech, of a scanning, rarely of a dysarthric character. (3) Disturbances of intelligence; these were absent in our cases, after the severe first stage. To these we may add: (4) Not infrequently exquisite spastic symptoms in the legs, more rarely in the arms, with corresponding gait and often greatly increased tendon reflexes; and (5) as a negative symptom the absence of all sensory disturbances, including the muscular sense, and of all disturbance of the sphincters. Westphal had already emphasized this point. As regards special details and variations in the course of the disease the reader is referred to the above-mentioned clinical histories and to the writings of Westphal and Leyden. It has been the custom—this is especially true of Leyden—to apply the term ataxia to the motor disorders of disseminated encephalomyelitis. This term should be employed with reserve, because the disorders are in reality very complicated and are composed essentially of two factors, which may also occur separately. For example, the gait may be exactly like the ataxic gait, as in Case III., except that the tottering is still more marked than in tabes; in other cases the gait is more paretic. It is often typically spastic, so that at every step there is an elevation upon the tips of the toes, and the tremor, which finally attacks the entire body, at first is composed solely of these contractions of the calf muscles. In the recumbent posture the tremor predominates in movements of the legs, but ataxic symptoms are by no means entirely absent. The symptom known as static ataxia is especially apt to be present. In our cases the motor disturbance of the arms consisted of almost pure, coarse intention tremor; the ataxic element was very slight. The latter predominated, on the other hand, in Westphal's cases. This is also true of the tremor of the head, which was exactly like that of multiple sclerosis. In brief, we find most frequently a mixture of typical tremor and ataxia, often also paresis. In other cases one of these symptoms may occur singly. The writer cannot refrain from pointing out that, as first noted by him, it is sometimes very difficult to distinguish ataxia and intention tremor from one another. As the myelitic foci, particularly those in the brain, may vary greatly in location in the individual cases of this disseminated form, the symptoms, as a matter of course,

will also vary. In some the tremor, in others the ataxia will predominate. Pure ataxia is rarer, as is shown by the absence of all disturbance of the feeling of position.

As already remarked, there was no disorder of intelligence in our cases, apart from those seen in the severe initial stage. This distinguishes them from progressive paralysis, in which extreme tremor, resembling intention tremor, also occurs. In severe cases the scanning speech may develop from complete motor aphasia.

The course and termination vary greatly. Thus in Case I. there was rapid and complete recovery; in the briefly reported case following pertussis, recovery occurred after a long time. In other cases (Case III.) certain symptoms remain permanently; this seems to be especially true of the speech disturbances. In a third series (Case II.) periods of improvement and relapse alternate. Death is rare, outside of the first stage in which an accurate diagnosis is very difficult, or it is the result of complications.

The clinical history, especially in the second stage, simulates that of acute multiple sclerosis. Even in multiple sclerosis there may be acute onset and exacerbations, but we usually have to deal here with individual symptoms. Multiple sclerosis is not attended by the violent onset of an affection of the entire nervous system. Nevertheless, Marie is inclined to assume a direct transition of acute disseminated sclerosis into multiple sclerosis. It must be remembered, however, that such a course has not been clinically observed and that, as Leyden remarks, disseminated encephalomyelitis has no tendency to further extension.

Diagnosis.

We will first discuss *acute* or subacute transverse myelitis. If the clinical history sketched above develops in an acute or subacute manner, it is especially characteristic in the grouping, succession, and gradual extension of the symptoms, and the diagnosis of disease of the spinal cord will be quite easy and comparatively safe. The only difficulties (but they are often insurmountable) arise when we attempt to make the diagnosis of a true inflammatory affection—of a myelitis in the stricter sense. Here the etiological factors are the most important in diagnosis. If an acute spinal paralysis develops after an infectious disease or poisoning—all the above-mentioned diseases and toxæmias and probably many others must be considered—then the inflammatory character of the disease is quite certain; and if there is no doubt of its localization in the spinal cord, we can only have to deal with a true myelitis. A difficulty in some cases resides in the differentiation of transverse myelitis from multiple neuritis, inas-

much as the latter is also especially apt to occur after infections and intoxications. Such differentiation may be impossible in some cases. This is evident when we remember that clinical observations show the frequent combination of myelitic and neuritic paresis, and that recent exact anatomical investigations have proven that the cord rarely is entirely intact in multiple neuritis. Differential diagnostic features may be presented by the entire clinical history and by special individual symptoms. In the first respect dorsal myelitis, for example, presents hardly any diagnostic difficulties. It is true that the tendon reflexes may also be exaggerated for a time in neuritis, but contracture of the legs does not occur. As the latter symptom is also found in cervical myelitis, this may be mistaken for a neuritis of the brachial plexus, but only so long as the myelitis is confined to the gray matter. Here pupillary and palpebral fissure symptoms would favor the diagnosis of myelitis. It is often difficult, if not impossible, to distinguish a neuritis of the lumbosacral plexus from a lumbar myelitis. Features common to both affections are the flaccid paralysis with atrophy and degeneration reaction of the muscles, absence of the tendon reflexes, symmetry of the paralyses, and perhaps their restriction to the lumbar or sacral plexuses. As a general thing the initial pains in neuritis are more persistent and violent than in myelitis, and tenderness of the peripheral nerves to pressure is not present in myelitis. On the other hand, anæsthesia is barely indicated in neuritis, bedsores do not form, and bladder disorders are extremely slight. The differential factors during the development and course of the disease are the following: while transverse myelitis often begins in an apoplectiform manner, neuritis develops subacutely with the exception of rare cases; in the latter affection the paralysis begins a considerable time after the irritative symptoms, especially the pains. The diagnosis is positive in favor of myelitis when the disease begins, for example, with paralysis of the bladder, which hardly ever occurs in neuritis. Recovery is extremely rare in myelitis; in multiple neuritis it is a frequent termination. Lumbar myelitis, which gives rise to especial difficulties in differential diagnosis, almost always has an unfavorable termination.

Hence if neuritis can be excluded in an acute or subacute paraplegic paralysis, and the etiological factors which enter into both diseases are present, the diagnosis of true myelitis may be made. The matter is entirely different and much more difficult when these characteristic and causal factors are wanting. Here the greatest difficulty is presented by the differentiation from hemorrhage and thrombotic softenings of the cord. In hemorrhage the diagnosis is still feasible. Extensive spontaneous hemorrhages, which resemble

cerebral apoplexy, are very rare in the cord. The most frequent are extensive traumatic hemorrhages confined to the gray matter. Here the history of the case facilitates the diagnosis of a hæmatomyelia or perhaps a hæmatorrhachis. In the latter event the pains and rigidity of the back exhibit a severity which is rare in myelitis.

Much more important, but also much more difficult, is the differentiation of acute myelitis from those vascular softenings which are due in rare cases to embolism, and more frequently to thrombosis. We have explained at sufficient length that these cases are frequent and that, in particular, the so-called acute syphilitic myelitis is usually of thrombotic origin. Thrombotic softening, dependent on vascular disease, may also develop subacutely, when small vessels are slowly occluded in succession. That the differentiation of these cases from acute myelitis is not possible according to present notions is apparent from the fact that the theory of myelitis is based almost exclusively on the observation of such cases. We believe, however, that a correct opinion will be reached if, in acute paraplegias of syphilitics, in which all prodromes, especially pains, have been absent, we suspect vascular softening. The termination will usually confirm the correctness of this view, because in these vascular cases inunctions will naturally prove as useless as in the analogous affection of the brain.

Lesions which compress the cord—caries, tumor of the spine, and intravertebral tumors—are mainly apt to be mistaken for chronic myelitis. But the sudden sinking-in of the spine in cases of caries and vertebral tumors, and the acute œdema in those of intravertebral tumors, may give rise suddenly to paraplegias at a time when the true nature of the primary disease is not clear. As a general thing, however, a deformity will be demonstrable in diseases of the spine, and tumors of the meninges will usually have been preceded by a protracted period of root symptoms.

It is hardly possible to mistake acute myelitis for meningitis, because clinically we scarcely ever find primary spinal meningitis, and tuberculous and purulent forms, although they extend to the cord, also involve the cerebral meninges and thus exclude the diagnosis of myelitis. Syphilitic meningomyelitis may occasionally extend acutely to the cord, but, as in tumors of the membranes, root symptoms have usually been present for a long time. The diagnosis will be decided by the history and the results of treatment.

Hysteria produces clinical pictures which are more apt to remind us of chronic myelitis, but in rare cases a symptom complex may develop acutely which may arouse the suspicion of myelitis—for example, a spastic paraplegia with contracture and increased reflexes. It would carry us too far to mention all the differential features.

Usually the etiological element in hysteria is psychical; the anæsthesia which may be present exhibits characteristic boundaries; the contractures are excessive while the tendon reflexes are not so pronounced; the clonus is not so protracted as in organic diseases; cystitis, decubitus, muscular atrophy, and degeneration reaction are wanting. Hence the diagnosis of acute transverse myelitis is to be made when etiological factors which favor inflammation attend the rapid development of paraplegic symptoms and multiple neuritis can be excluded. If these etiological factors are lacking, the differentiation from acute compression is usually easy; but that from vascular softenings is difficult and often impossible. In syphilis acute vascular softening of the cord is by far more frequent.

Everything has been said above concerning the segment diagnosis. It may here be mentioned that when the disease involves only a part of the transverse section, the general as well as the segment diagnosis of myelitis becomes difficult. The clinical pictures of other specific forms of cord inflammation, for example anterior poliomyelitis, or obscure symptom complexes will then be produced. As a matter of course we cannot discuss all the possibilities.

Before making a diagnosis of *chronic* transverse myelitis, which is an extremely rare disease, all other possibilities must be excluded. The diagnosis of a chronic myelitis is most plausible when it follows a disease which is positively known as an acute spinal inflammation. If this is not true, we must exclude, on account of their relative frequency, the chronic compressions of the cord, the most frequent of which is that due to spinal caries. As a matter of course only those cases need be considered in which the morbid symptoms on the part of the spine are slow in developing. Such cases are by no means rare and then nothing remains except to make a provisional diagnosis of chronic myelitis. This is also true of the much rarer compression by tumors of the vertebræ and spinal meninges and by pachymeningitis—especially hypertrophic cervical pachymeningitis. All these diseases will hardly be mistaken for myelitis when they run a so-called classical course, but they may deviate so much from their characteristic symptoms that it is impossible for a long time to distinguish them from a simple chronic myelitis. In such doubtful cases it is always important to examine the spine repeatedly for deformity or local tenderness. For an account of the more special symptoms we refer to the sections on caries of the spine and spinal tumors.

Among the diseases of the spinal cord itself the differential diagnosis must take into consideration syringomyelia, so-called combined system diseases, and, above all, multiple sclerosis. Syringomyelia, which was formerly regarded as a periependymal or cavitary mye-

litis, can be mistaken for myelitis only in cases in which (for example, when the cavity is located in the dorsal cord) the clinical symptoms consist in the main of a spastic spinal paralysis, and the characteristic sensory disturbances are not distinct. The fact that some cases of combined system disease cannot be distinguished from chronic myelitis is evident from the statement of Leyden and Goldscheider that many cases are really instances of chronic myelitis; their symptoms often exhibit the characteristics of a diffuse disease of the cord. But generally, as Oppenheim has emphasized, multiple sclerosis is wrongly regarded as a chronic myelitis. Every observer, on careful investigation, finds an increasing number of cases of multiple sclerosis in which the first symptom developed acutely, and myelitis is then readily excluded, especially when this first symptom, as so often happens, was an inflammation of the optic nerve. But this does not happen in all cases. An insidious slow onset and progress of all the symptoms are also frequent in multiple sclerosis. A confusion with chronic myelitis arises in cases in which, while foci are present in the brain, they do not give rise to distinct symptoms, or when (as happens in very rare cases) multiple sclerosis appears anatomically in the shape of a diffuse affection which is confined to the spinal cord. In all these doubtful cases we shall often hit the nail on the head by making a diagnosis of multiple sclerosis rather than of myelitis. Chronic syphilitic meningomyelitis may be distinguished from simple chronic myelitis by the previous history and the final outcome. In these cases the disease is very rarely confined to the spinal cord and still more rarely to one spot in the cord. The cases are much more frequent in which the symptoms indicate manifold foci, and usually in the brain as well as the cord.

Formerly chronic multiple neuritis was often mistaken for chronic myelitis; the reports of cases of chronic myelitis terminating in recovery probably depend in part upon this mistake. The differential points in the diagnosis of acute neuritis and myelitis have been given above, and *mutatis mutandis* they may be applied to chronic cases. We have already said that hysteria may also simulate a chronic myelitis. Here the differentiation may be difficult even for the expert; the important factors have been mentioned under the heading of acute myelitis.

Landry's paralysis has such characteristic symptoms that its recognition does not offer the slightest difficulty. It is difficult, however, and often impossible to distinguish the peripheral form of this paralysis from the central variety. The termination in recovery almost always appears to be decisive in favor of the peripheral form. In addition, a slow course, severe pains, and, above all, tenderness

of the nerves on pressure favor the diagnosis of the latter variety; this is also true of atrophy and distinct electrical disturbances of the muscles. After alcoholic excesses the peripheral form is undoubtedly more frequent than the central.

Disseminated encephalomyelitis also exhibits such a well-defined character in its etiology, symptoms, and course that the diagnosis may be called an easy one. In the first stage, however, with its severe general and mental symptoms, a positive diagnosis may be impossible. If we are brought face to face with the fully developed symptoms of the second stage and know nothing of their mode of onset, it is naturally possible to mistake the disease for multiple sclerosis. If Marie were correct in his opinion that these acute forms often pass into true multiple sclerosis, this would not be a mistake, but such a transition has not been clinically observed. Until this is done it is better to regard disseminated encephalomyelitis as a special disease which has no tendency to progress and is distinguished from multiple sclerosis by the sudden onset of all the symptoms at once, following directly an infection or intoxication.

Cases of disseminated encephalomyelitis with impaired intelligence may be mistaken for general paralysis. In the latter affection the tremor may be as pronounced as in the former; in not a few cases paresis is mistaken for multiple sclerosis. In encephalomyelitis the dementia is never progressive; the absence of syphilis also weighs heavily against the diagnosis of general paralysis.

The extremely rare ataxia (pseudotabes peripherica) which is due to neuritis, can hardly be mistaken for the central variety. As complete restriction of peripheral neuritis to the sensory nerves hardly ever occurs, careful observation will disclose muscular paralyses with atrophy and electrical disturbances. Furthermore, the tendon reflexes are absent; the patients suffer from pains and anæsthesia. All these symptoms are absent in disseminated encephalomyelitis. In peripheral pseudotabes the disturbances in movement consist of pure ataxia; intention tremor is wanting. A feature common to both diseases is their occurrence after infectious diseases and intoxications.

Prognosis.

The prognosis of *acute* transverse myelitis is very serious and always doubtful. In many cases, perhaps in the majority, death is the inevitable termination. This may occur in the acute stage, soon after the beginning of the disease or only after prolonged suffering. The former event occurs particularly in the cases known as myelitis acutissima in which the disease extends rapidly over large

parts of the transverse and longitudinal sections of the cord. Cases in which the paralysis runs an ascending course are especially dangerous. If death does not ensue, the most varied terminations are possible. Most frequently there is considerable improvement, but this remains imperfect. It is rare that the disturbance of function remains the same as at the height of the disease, and still rarer is the termination in complete recovery. More special data concerning the prognosis of individual cases can hardly be furnished, indeed it is scarcely possible to make a general prognosis in regard to life or complete or incomplete restoration to health. The following general viewpoints may serve as a guide in the prognosis: (1) The rapidity of the development and extension of the disease; (2) the level of the lesion; (3) the causal factor; (4) the absence or presence of severe complications, particularly decubitus. As a matter of course these four factors cannot be separated sharply because they partly involve one another. Let us examine these conditions a little more closely. The acute onset of the disease has a bad prognostic significance, when associated with rapid and diffuse extension along the transverse and longitudinal sections. But if, on the other hand, the symptoms indicate the affection of a small section of the cord, especially an incomplete transverse section, the prognosis is relatively favorable when the inflammation begins very acutely. In such cases the process usually comes rapidly to a standstill, while the cessation of the progress can never be told when it spreads slowly and more subacutely. The prognosis becomes much more serious, whatever the level of the disease, if the entire transverse section is involved. In these cases the sensory disturbance below the lesion is complete, so that the danger of bedsores is much greater and the disorders of the bladder and rectum are also especially severe.

In regard to the second factor, viz., the level of the disease, the most favorable prognosis is offered by cases of circumscribed and not total transverse dorsal myelitis. In these cases, which are fortunately the most frequent, the improvement is often very marked. So-called spastic paresis is generally left over; the paresis and spasm are often very slight and only the permanent clonus of the tendon reflexes indicates the previous existence of myelitis. Usually, however, very slight vesical symptoms are also present.

In lumbar myelitis the vesical and rectal disorders are much more pronounced and hence cystitis, with its dangerous sequelæ, and decubitus are much more frequent. Death usually occurs after a comparatively short time, and in the rare cases in which life is preserved the paralysis remains flaccid so that the ability to walk is not restored, as in dorsal myelitis.

The most dangerous form is cervical myelitis. If it extends to the phrenic nucleus and is, in a measure, transverse, so that the thoracic respiratory muscles are paralyzed, death is inevitable. If the patient escapes with life, atrophic paralyses persist in the arms in addition to spastic paralysis of the legs, *i.e.*, the ability to work is almost entirely abolished.

Important prognostic data are also supplied by the etiology. Pick had remarked and Oppenheim strongly emphasizes the fact that cases with an undoubted infectious etiology offer, on the whole, a relatively favorable prognosis. He refers particularly to the good prospects of recovery in gonorrhœal myelitis (this is frequently a meningomyelitis). *A priori* it is not improbable that the cases of myelitis will correspond to the varying degree of danger of the primary infections, the level and extent of the disease also being taken into consideration. Thus, it is easily understood that myelitis as the result of tuberculosis has a very serious prognosis. To the comparatively favorable course of acute transverse myelitis after infections is probably due the fact that cases attended with very severe pains usually permit a favorable prognosis. The pains indicate a combination with neuritis and the latter is especially frequent after infectious diseases.

As a matter of course the age and general condition of the patient at the beginning of the disease exert a very considerable influence on the prognosis. The tendency to a fatal termination is least during childhood. Very great importance attaches to satisfactory nursing, especially when death does not occur during the first stage, and a long, severe spell of sickness follows. The third important prognostic factor, viz., the complications (bedsores and cystitis) depends mainly upon the nursing. The latter determines whether these complications will develop or, at least, whether they will attain a degree which threatens life.

The prognosis of *chronic* myelitis is more serious than that of the acute form, because the disease almost always advances uninterruptedly and finally destroys vital centres or leads to death from marasmus. A standstill occasionally occurs and then the prognosis depends upon the amount of destruction in the cord. Termination in recovery should always arouse the suspicion of a wrong diagnosis, particularly of confusion with neuritis or with compression of the cord in cases of spinal caries.

Landry's paralysis with central localization almost certainly proves fatal. The diagnosis is a sentence of death. We should give a cautious prognosis, however, because the differentiation of central from peripheral ascending paralysis is not always possible and recovery often occurs in neuritis.

On the other hand the prognosis of acute *disseminated encephalomyelitis* is very favorable. Here we must consider the usually distinct infectious etiology and also the fact that the disease generally occurs in youth, often in childhood. Death occurs, if at all, in the first stage, which is obscure in regard to diagnosis. Complete or nearly complete recovery is frequent, but there is a tendency to relapses. Here also the prognosis of the nervous disease may correspond to that of the primary infectious disease. I observe complete recovery after varicella and pertussis, and partial recovery after scarlatina. As a matter of course, the extension of the disease must also be considered. The prognosis would be different if these cases often passed into multiple sclerosis, but this sequence has not yet been proven.

Treatment.

The treatment of *acute* transverse myelitis naturally is divided into two sections: (1) The treatment of the disease during its development and at its height; and (2) the treatment of the period of convalescence and of the permanent sequelæ. As the treatment of the second half of acute transverse myelitis is hardly different from that of the primarily chronic form, it will be discussed later in describing the treatment of the latter affection, and we shall confine ourselves for the present to the consideration of intervention in the first stage of acute transverse myelitis.

Is there a prophylaxis of acute myelitis? This can only proceed from a knowledge of the etiological factors. We know that the disease occurs mainly after infectious diseases and intoxications. The prophylaxis would then be directed towards minimizing these two causes as much as possible; in regard to intoxications, this subject possesses intimate relations with the hygiene of trades and foods. Moreover, as the possibility of an invasion of the spinal cord is present in almost all infectious diseases, the greatest caution must be exercised in these diseases. For example, the patients must be warned against bodily exertion during convalescence, inasmuch as this may possibly convert the cord into a *locus minoris resistentiæ*.

Formerly the abortive treatment of myelitis was much discussed, and Erb and Gowers still mention it. As a matter of course it is impossible in very acute cases, while in those which begin more subacutely it may possibly be useful if employed very early. But the question arises whether any remedies can prevent a myelitis which is already developing, and then again who would venture to make a positive diagnosis of myelitis at such an early stage? Hence we possess no means of judging of the value of the treatment adopted,

and, if the latter should be followed by recovery, we would often be justified in excluding myelitis. For all these reasons we may employ in abortive treatment only such measures as cannot, at least, do any harm. The best measure would be diaphoresis with hot packs, as we are justified in assuming that a favorable effect may be produced by vigorous diaphoresis in infectious diseases.

At the height of the disease the main object of the physician should be to secure the best possible care of the patient. This is not a trifling matter. By a careful supervision of all details, even those of an apparently trifling character, the physician can often do much service, and above all he can prevent harm. From the very beginning of the disease the patient requires rest in bed; at the height of the disease this follows of itself. The rest in bed must be carried out strictly, and the patient may not be removed even to attend to the wants of nature. The arrangement of the bed itself is very important. Upon a spring mattress is placed a hair mattress which, for convenience of removal, should be made of several parts. Upon this is placed a linen sheet, and then a large waterproof cloth. The middle and lower parts of the patient's trunk are placed from the start upon a large water bag, over which is spread a linen cloth. The pillows should also be made of horsehair. Folds in the bedding should be carefully avoided. When possible two beds should be kept in readiness, so that he can be placed in the second one when the first one is made up or when it has been soiled. It is also very comforting to the patient to be placed in a fresh bed at nightfall. If two rooms are at our disposal the beds should be placed on castors so that they may be easily rolled from one room into the other. The patient should not constantly occupy the same position in bed; he should be changed from the back to one side or the other and, if possible, occasionally to the abdomen. These recommendations alone will often suffice to avert the greatest danger to the life of the patient, viz., bedsores; in addition, the most scrupulous cleanliness is necessary. The cleanliness is interfered with to the greatest extent by the frequent disturbances of the vesical functions. More rarely the rectal disturbances act in this way. The most careful attention is necessary because, in the most severe cases, the patient himself does not notice the soiling; when this takes place the patient and the bed should be cleaned at once, if possible. This is best done by keeping a bathtub in the sick-room. The patient is placed in the tub, washed, and the bed arranged at the same time. The soiling of the bed with urine may often be kept within certain limits. If there is weakness of the detrusor, the catheter should be used at the proper time when there is complete retention of urine. This also holds good in incon-

tinence because the bladder contains a good deal of urine in cases of ischuria paradoxa, and after its evacuation the incontinence often ceases for a time. In the severest cases of vesical paralysis when the sphincter vesicæ can retain even small amounts of urine for a brief period only and the urine is escaping almost constantly, artificial urinals, into which the penis is placed, may be employed in males in order to prevent continued wetting of the bed. It must be remembered, however, that in such cases decubitus develops not infrequently upon the glans of the anæsthetic penis or œdematous prepuce. These accidents should be guarded against by placing cotton between the glans and urinal and frequently changing the cotton. Good urinals have not yet been constructed for women. It is best to place between their thighs large pledgets of cotton or pillows of moss, and to renew them frequently.

Soiling with fæces generally occurs only every few days, because there is usually constipation and great dryness of the fæces. It may often be prevented entirely by the regular removal of the hard fæcal masses either manually or by means of injections.

Alcoholic lotions for the back are very popular among the laity. At all events they do no harm, especially when the patient is otherwise kept clean.

It must be conceded, however, that decubitus cannot always be prevented, despite the careful employment of these measures. When decubitus has developed, it is best to apply borated or iodoform ointment several times a day and also after each soiling, until the gangrenous parts have been exfoliated; when the granulations are exposed nitrate of silver ointment may be used. With careful attention in cases in which the disease in general has a tendency to improve, we may often cure deep bedsores or at least prevent their extension. We shall then not be apt to observe the very grave cases, for example, those attended with erosion of the vertebral column.

Next to decubitus the life of a patient suffering from myelitis is most threatened by cystitis and its sequelæ, pyelitis, nephritis, and uræmia. If a patient suffering from ischuria paradoxa and incontinence of urine is not properly cared for, cystitis will inevitably develop. The urine must be evacuated artificially, usually with the catheter. Everything depends upon the most scrupulous cleanliness of this instrument. Metallic catheters should be passed through an alcohol flame immediately before they are used; elastic catheters should be kept constantly in three-per-cent. carbolic-acid solution. Irrigation of the anterior part of the urethra is also indicated before catheterization, in order that infectious germs may not be pushed from this part into the bladder. We believe, however, that in long-

continued paralysis of the bladder, when catheterization is necessary, cystitis can hardly be prevented. When the bladder can be expressed this manipulation is accordingly indicated.

A cystitis which has developed may be relieved by irrigations of the bladder with four-per-cent. boracic acid. Mineral waters, such as those of Wildungen, Bilin, and Vichy, may also be given, especially if nephritis is threatening. On account of the increase in the amount of urine the use of these waters will still further increase the difficulties in nursing.

The patient should be well nourished, but as a matter of course we must avoid food which is difficult of digestion or excessive in amount. On account of the cystitis, beer, wines, and spices should be avoided.

Only the well-to-do can be cared for properly at home; other patients should, in their own interests, seek admission to a hospital.

In regard to more active therapeutic procedures, the first recommendation is *nil nocere*. In former times, particularly in France, much reliance was placed upon so-called antiphlogosis. The inflammation was to be relieved by the application of ice to the spine, of Chapman's ice-bag, or by derivative measures, inunctions of gray ointment or application of iodine, cups, or the vigorous use of the actual cautery. The effect of all these measures is problematical, and the more vigorous ones among them must certainly be discarded because they directly invite the danger of decubitus. We would be most inclined to use inunctions of gray ointment, especially if there were any suspicion of syphilitic meningomyelitis. We have already expressed our opinion concerning the use of diaphoresis. As a matter of course, this may be employed not alone as an abortive measure but also at the height of the disease.

Not much can be expected from medicinal remedies in myelitis, and, unless there are positive indications for quinine, it is best to discard them entirely. If the slightest suspicion of syphilis arises, potassium iodide or, better still, mercury must at once be administered vigorously. It must be remembered, however, that "acute syphilitic myelitis" is almost always a vascular softening, and that restoration of the parts destroyed by the softening cannot be attained, at the most we can only prevent the further extension of the disease of the vessels.

In the acute stage hot baths, especially steam baths, should be avoided; warm baths may act favorably. The employment of electricity in the acute stage of myelitis is a useless annoyance.

The pains must sometimes be relieved symptomatically, and since they do not last long in myelitis we need not hesitate to use mor-

phine. Sodium salicylate may also be tried. The muscular twitchings in the legs are often very distressing and can hardly be relieved. Warm baths perhaps are most useful. Hypnotics must sometimes be employed.

It is evident that the prospects for therapeutic successes in acute myelitis are not very promising. Complete recovery occurs in rare cases and more frequently there is very considerable improvement, but we would be inclined to attribute these favorable terminations rather to the *vis medicatrix naturæ*. We are satisfied when careful attention places the patient in the most favorable position for the healing action of nature. As a matter of course, still less can be expected from the treatment of chronic myelitis. It is characteristic of this disease to progress uninterruptedly and finally to prove fatal by the destruction of vital centres. Nevertheless, we may not idly fold our arms but must employ all measures which may bring any relief to the patient or which offer any hope of effecting an arrest of the disease.

In the treatment of *chronic* myelitis as well as the convalescent stage of acute myelitis, we lay chief stress on a number of physical methods, balneotherapy, hydrotherapy in the stricter sense, and electrotheraphy, the latter in combination with gymnastics and massage. We may remark here that the credit enjoyed by these methods of treatment must be attributed to the results obtained in the convalescent stages of acute myelitis rather than in chronic myelitis proper. Erb has made the most extensive and critical study of balneotherapy and hydrotherapy in myelitis, and we merely reproduce here his statements. He advises against the use of indifferent thermal waters; those which have a high temperature are directly dangerous, particularly in recent cases and in old ones which have a tendency to exacerbations. Much more favorable are the results with cool, carbonated baths, for example, in Oeynhausen and Nauheim. These baths may not be too cool nor their duration too long and the amount of carbonic acid should not be too great. Sea-bathing is to be recommended only for very vigorous individuals when recovery is practically complete. Erb has a very favorable opinion concerning hydrotherapeutic measures in myelitis. Only mild forms may be used—simple packs, half baths, and sitz baths. Very low temperatures, vigorous douches, and protracted applications are to be avoided. The water cure should not be continued too long without interruption. It is preferable to repeat it after a while. An essential element in the water cure is the increase in the appetite and the stay in the open air. In view of a very disagreeable personal experience, we regard steam baths as directly dangerous in chronic myelitis.

The hopes which were formerly placed in the electrical treatment of chronic myelitis were greatly exaggerated. To-day there are probably very few neurologists who believe in the direct action of electricity upon the diseased spinal cord. Nevertheless, direct galvanization of the spine is the best method, because it is at all events innocuous. We must advise against faradization of the paralyzed parts, if they are contractured, because this may give rise to annoying spasms. In like manner galvanization should be applied very cautiously upon anæsthetic parts, on account of the possibility of producing excoriations. The flaccid paralyses of lumbar myelitis may be directly treated with electricity—preferably with galvanism—but we are hardly justified in attributing favorable results to this treatment. A favorable action upon weakness of the bladder is sometimes exerted by vigorous direct galvanization of the vesical region, with interruptions and changes of polarity.

Massage and mild gymnastics are advisable when, as happens chiefly in dorsal myelitis, the patient is again able to walk and suffers only from spastic paresis. Great caution is necessary, and above all exhaustion must be avoided, because this is apt to produce a relapse. Short walks with frequent periods of rest are the form of gymnastics best adapted to the patient, from a mental as well as physical point of view. Mercury and potassium iodide may be employed whenever the slightest indication exists. A trial of nitrate of silver is also allowable. In the convalescent period of acute myelitis, tonics, such as iron, quinine, cod-liver oil, and arsenic, are indicated, and particularly nourishing food. The patient should be in the open air as much as possible. The treatment can best be carried on by a trip to a well-conducted sanatorium in a mountainous region.

Complications, such as decubitus and cystitis, are treated in the same way as in acute myelitis. We must be more careful in the administration of morphine to relieve pain, because as a matter of course the danger of producing morphinism is greater in chronic cases.

In *Landry's paralysis* of a central character vigorous diaphoresis is indicated. On account of the danger of "schluck pneumonia," special consideration must be paid to the disorders of deglutition.

Diaphoresis is also indicated in the acute stage of *encephalomyelitis*. The proper nursing of the patient is sometimes rendered difficult in this stage on account of the delirium. The severe choreic movements may give rise to injury, while the danger of decubitus is less because anæsthesia is generally absent. In the cases which rapidly run a favorable course, nothing need be done at a later

lection, which might eventually lead to the production of distant abscesses by metastasis; it will become more probable when other symptoms of pyæmia are also present, and it becomes most probable when the primary purulent focus is a putrid bronchitis or perhaps a purulent cystitis, as these two affections seem to have a particular tendency to metastases in the nervous tissues. As in abscess of the brain, so also here the main point in the diagnosis is the demonstration of the primary abscess.

The *prognosis* is absolutely bad. Death always occurs. In most cases the primary disease is fatal in itself, the abscess of the spinal cord and the purulent meningitis serve only to hasten the end.

There is no *treatment* of spinal-cord abscess that will avail. In the case of a metastatic abscess we might possibly evacuate the pus, but when such a diagnosis is possible, an incurable primary lesion is usually present, as well as other non-operable metastatic abscesses. Furthermore, as has already been said, the localization of an abscess in the spinal cord is peculiarly difficult, and the metastases in these cases especially are frequently multiple. Even in the unlikely event of a complete evacuation of the spinal abscess, any marked improvement in the symptoms could scarcely be hoped for.

SYPHILIS OF THE SPINAL CORD.

Syphilis, as it occurs in connection with the spinal cord, may be either extraspinal or intraspinal. The extraspinal origin is the more rare. It presents itself in the form of a syphilitic disease of the bones, generally a periostitis gummosa of the spine, followed by caries and necrosis of the bone, and finally by involvement of the spinal meninges and of the cord itself.

Intraspinal syphilis has various forms. The chief type is syphilitic meningitis or meningomyelitis spinalis. The disease begins in the dura mater with the formation of syphilitic granulation tissue and specific disease of the vessel walls. In its further course the affection extends laterally through the spinal cord and causes a transverse myelitis. But this latter form, transverse myelitis, may arise primarily, *i.e.*, without passing through the meninges. It is then probably due to specific vascular changes.

Isolated syphilitic affections of individual spinal tracts are doubtful. A special form of lateral sclerosis, which is said to depend upon a primary syphilitic infection, has been described by Erb. And, finally, the syphilitic infection may find expression in the form of gummata of the spinal cord.

All of these affections are directly connected with the infection of

syphilis, and may consequently be regarded as direct sequelæ of it. Not so with the indirect sequels of syphilis of the cord, which probably depend rather upon the morbid products of syphilis, and are therefore with propriety called metasyphilitic processes. The type of these is tabes dorsalis.

Syphilis of the spinal cord may also appear as a part of general syphilis of the central nervous system, lues cerebrospinalis.

SYRINGOMYELIA.

There is probably no affection of the spinal cord which has been more thoroughly studied of late years than syringomyelia, and our knowledge of this singular disease has been correspondingly increased. Although as recently as 1878 Erb could say in his textbook of diseases of the spinal cord that "at present there are no means of making the diagnosis of syringomyelia during the life of the patient, we can only make vague suppositions regarding it," at the present time a series of well-defined symptoms enables us to diagnosticate the disease with certainty.

Etiology.

The etiology of syringomyelia is still absolutely unknown. In the majority of cases there is probably an anomalous embryonic development, which in later years results in syringomyelia uninfluenced by external causes. Occupation has nothing to do with it. Overexertion, especially of the hands, is also of very doubtful etiological importance. Yet trauma is not to be absolutely rejected as an accessory cause, although it can only develop the tendency to the disease which is already present—it cannot cause the disease itself. Falls and blows upon the back, and, recently, injuries of the hands, and especially of the fingers, often of a quite trivial nature—*e.g.*, punctures by small splinters of wood or metal—have been reported as exciting causes. Occasionally infectious diseases seem to initiate the affection. Thus, cases have been described in which the disease developed as a sequel of typhoid fever, scarlet fever, measles, articular rheumatism, intermittent fever, pneumonia, or pleurisy. Finally, it should also be mentioned that the first symptoms often appear after childbirth, although we are not justified in inferring any etiological connection of this process with the disease. Chronic alcoholism and syphilis, the two factors which play so prominent a part in the causation of other nervous diseases, need hardly be considered in the case of syringomyelia.

An inherited tendency to the disease seems never to be present. With respect to the sex of the patients, the majority appear to be males, but statistics naturally vary somewhat according to the class of patients observed by each author. The age in which the disease first appears is most frequently between the eleventh and the thirtieth year. Cases are very rare after the sixtieth year. Schlesinger found the age in one hundred and ninety cases to be as follows:

	Males.	Females.
1 to 10 years	4	1
11 to 20 "	36	8
21 to 30 "	53	25
31 to 40 "	30	12
41 to 50 "	4	7
51 to 60 "	3	3
Above 60 years	3	1
	133	57

In women the disease appears to begin late in life more frequently than in men.

Pathological Anatomy.

Anatomically, syringomyelia is a formation of cavities within the spinal cord, which cause the organ to resemble a pipe (σῦριγξ), whence the name. This process of the formation of cavities may affect the central canal, resulting in a progressive widening of the latter—the disease then being called hydromyelia; or the cavities are formed independently of the central canal at the outset—syringomyelia in the narrower sense of the word. Cases of true hydromyelia will not be further considered, since they present either no clinical symptoms or such as are absolutely incapable of diagnosis.

Syringomyelia in the narrower sense of the word consists in the formation of cavities, which develop with especial frequency behind the central canal in the gray commissure, and from this point extend laterally in an irregular way. The cavities involve by preference the posterior cornua and columns, and occasionally attack also the anterior cornua in the anterior columns, but almost never the lateral columns. The form and size of the individual cavities are as irregular as their distribution; the transverse section is therefore quite unsymmetrical. The cavities are of the most various forms, sometimes circular, sometimes oval, sometimes a long fissure. Their breadth varies as greatly. It is generally between 1 and 10 mm., but there are cavities in which one could insert the tip of the little finger. The cavities are generally filled with a turbid fluid, so that before the spinal cord is incised a true fluctuation can be produced. They are

always separated from the healthy tissue by a firm, hard membrane. Their interior is lined with epithelium. Occasionally it can be demonstrated that several small cavities have united to form a single larger cavity; not infrequently, too, some of the cavities become connected with the central canal.

The whole spinal cord and even the medulla oblongata may be attacked by the disease. Its favorite location is, however, the cervical cord; the process almost invariably begins there.

What is the origin of these cavities? Without discussing the differences of opinion of the various authorities, we may state the result of such investigations as have been made up to the present time to be that the cavities may originate in either of two ways: 1. From a congenital malformation of the central canal; 2. From proliferation and subsequent destruction of the neuroglia. The latter condition is called gliosis or gliomatosis. Occasionally both modes of origin are combined.

There are certainly cases which originate from a simple hydromyelia, *i.e.*, from an embryonic dilatation of the central canal, in consequence of disturbances in the process of its obliteration or in consequence of a constriction of the cord posterior to the central canal.

In other cases, the primary factor is a proliferation of the glia, which proceeds generally from the vicinity of the central canal, but which may also begin in the posterior cornua and occasionally in the white matter. Perhaps this is a process of fœtal origin, fœtal nests of glia cells persisting and being stimulated to proliferate by some secondary irritation; perhaps, as other authors think, the proliferation may originate also in the normal glia tissue. The view is also held that a proliferation of ependyma cells proceeds from the central canal and thus forms the basis of the process. However that may be, a proliferation, a gliosis, is developed in the glia. From this a cavity is formed through the destruction of portions of the tumor as the result of retrograde metamorphosis. The details of this process are still a subject of controversy, especially as to the question whether it is to be regarded as inflammatory or neoplastic.

As secondary changes in the nerve tissues consequent to the gliosis are found degenerations of the nerve fibres, degenerations and atrophy of the ganglion cells of the gray matter as well as of the anterior and the posterior cornua, and finally also vascular changes in the form of thickening and hyaline degeneration of the walls of the blood-vessels. Hemorrhages may also be found in the gliomatous tissue.

If, however, this tissue does not form cavities through retrograde metamorphosis, it grows steadily and assumes finally the character

of a tumor, as a result of which the whole spinal cord may appear considerably increased in volume. Upon transverse section there is then seen a solid tumor, generally surrounding the central canal, but occasionally demonstrable in other regions, especially the posterior cornua, which, precisely like the cavities, extend vertically through the cord to a varying distance. This condition is called central gliosis or gliomatosis. The formation of cavities and gliomatosis, then, represents the two terminal stages of gliosis. They may be found united in one spinal cord; there may be present a solid gliomatosis, which in one or more places has developed cavities.

Symptoms.

That which is most characteristic in the complexus of symptoms of syringomyelia is the combination of muscular atrophy with sensory and trophic disturbances. There is a series of other symptoms, but these must be regarded as secondary and comparatively unimportant.

We will first describe the individual symptoms, without reference to the order of their appearance.

The motor affections present themselves especially in the form of muscular atrophies. These are the result of the compression which the anterior cornua undergo through the growth of the cavities, but are also caused by the involvement of the gray anterior cornua themselves in the process of cavity formation, and finally by the previously mentioned secondary degeneration of the ganglia of the anterior cornua. The atrophy of the muscles, which may become extreme, is therefore always degenerative. Different electrical reactions are found, according to the intensity of the process. In case of a simple compression of the anterior cornua, either no change or at most a trivial quantitative diminution in the electrical excitability can be demonstrated. If the anterior cornua have become involved in the glia proliferation, or if they have already undergone secondary degeneration, their trophic influence over nerve and muscle is abolished, and the reaction of degeneration can be obtained from the muscle fibres which depend upon these ganglionic cells, *i.e.*, sluggish galvanic contraction with reversal of the contraction formula and abolition of faradic excitability. When through atrophy of the anterior cornua the muscle has become completely atrophied and consists only of fat and connective tissue, its electrical excitability will be lost for both currents.

The electrical examination of the atrophied muscles demands much practice and patience. Since fibres which have already degen-

erated often lie side by side with those which still react normally, the muscle must be gone over millimetre by millimetre, in order to find the reaction of degeneration.

In correspondence with the origination of the anatomical lesions in the cervical cord, the atrophy begins almost always in the hands, and especially in the muscles supplied by the ulnar nerve, involving subsequently the regions to which the median is distributed. Finally, all the muscles of the hand become atrophied, and the familiar clawed hand, the *main en griffe*, is produced, the preponderance of the flexors and extensors of the forearm resulting in abnormalities of position of the phalanges. Fibrillary twitchings are very frequently seen in the atrophied muscles. The atrophy may be more marked in one hand than in the other; at the beginning of the disease it may even be confined to one hand. As the disease progresses, the process may remain limited to the hands, or it may spread more widely. In the latter case the muscles of the forearm and lower part of the arm are generally passed over, and the muscles about the shoulder, especially the deltoid, are next implicated (Duchenne-Aran type). The other shoulder muscles are then attacked, and after them the muscles of the lower arm. If the disease still progresses, the muscles of the back are next involved, then the intercostals and the abdominal muscles; and finally, although this is extremely rare, the muscles of the lower extremities. Here the calf muscles, the glutei, the extensors of the leg, and very often the vastus, are most atrophied. And here, too, from the preponderance of the antagonists, deformed positions of the feet are met with.

Deviations from this typical course of the atrophy may occur, in that occasionally the affection may begin in the dorsal or lumbar cord, in which case the lower extremities are first and most severely affected, while the upper are involved at a much later period, and may even escape entirely. Exceptionally, when the disease is located in the cervical cord, the atrophy may begin about the shoulder and pass to the hand muscles subsequently (humeroscapular type).

The atrophy of the musculature is not always distributed symmetrically in the two halves of the body; there are even cases in which an absolutely one-sided atrophy is present (gliosis unilateralis).

In all cases in which the lower extremities are not themselves the seat of the disease they are indirectly implicated, since the so-called complexus of spastic symptoms develops in them—severe active and passive spasms of the muscles, with contractures and greatly increased patellar reflexes, occasionally also clonus of the feet. True ataxia is also often present. The question how this complexus of symptoms is to be explained needs further investigation. It has

been maintained that it arises from the secondary degeneration of the lateral pyramidal tracts, which frequently develops when the disease is located in the cervical cord. Others have expressed the opinion that simply the compression of these tracts by the gliosis or gliomatosis suffices to produce spastic phenomena.

With regard to the manifestations of motor irritation, the fibrillary twitchings of the atrophied musculature have already been mentioned. Slight tremor of the hands, which sometimes suggests intention tremor, local cramps of individual groups of muscles, and generalized convulsions involving considerable portions of the body have been reported.

The disturbance of sensibility which is characteristic of syringomyelia is the so-called partial paralysis of sensation, the discovery of which forms an epoch in the history of syringomyelia. It cannot, of course, be affirmed that this complexus of symptoms is found exclusively in syringomyelia, but it occurs in connection with the other symptoms only in this disease. The details will be considered under the head of differential diagnosis. Partial paralysis of sensation consists in this, that while the tactile sensations are perfectly normal or at most slightly lessened, the sensation of pain is much diminished and that of temperature appears to be quite abolished. This paralysis of sensation is never confined to the course of single nerves, but appears in segments corresponding to the divisions of the spinal cord—in an arm, in one-half of the trunk, in one leg. Its favorite location is the extremities and the upper part of the trunk. In general, it may be said that it pursues the same course as the atrophy.

In greater detail, these disturbances of sensibility present the following picture: The sense of touch is completely normal in almost all cases; contact with the finger tip, a brush, the head of a pin, is normally perceived and properly localized. There are, it is true, cases in which a more or less considerable diminution of the sense of touch is present, but they are very rare. In syringomyelia the sense of pain always undergoes a more or less well-marked diminution, which may become complete analgesia. The patients no longer feel pin pricks and faradic currents upon the skin as pain, and in the severe cases do not feel them at all. Extensive surgical operations may occasionally be performed upon them without pain. The sense of pain of the deeper parts may also be impaired, so that, for example, fractures of the bone are no longer felt to be painful.

The impairment of the sense of pain begins regularly in the fingers and the hand, and travels upwards to finally extend over the trunk. It may occasionally be unilateral only. From the arms it

may extend still farther upwards and involve the distribution of the trigeminus in the face.

The temperature sense, *i.e.*, the ability to distinguish differences of temperature, is totally abolished in most cases. Hence the following very characteristic feature of the disease: These disturbances of sensation are wont to appear first upon the fingers, and the patients may consequently receive burns upon them which they do not perceive, and only learn of from the resulting lesions; the hands and fingers, accordingly, present many scars as the result of such accidents. From the hands the disturbances of the temperature sense extend to the trunk. Objective examination shows that the patients in the parts affected either do not distinguish warmth and cold at all and in each case perceive only the contact of the warm or cold object, or that perverted sensations are present; warmth is felt as cold, and *vice versa*.

The mucous membranes also often participate in the disturbances of the temperature sense, so that the patients may eat too hot food without hesitation.

Besides these objectively demonstrable disturbances of sensibility, there are also subjective abnormalities. They consist of pains and paræsthesiæ, both especially localized in the hands and arms, and both often the first symptom of the disease. The pains are tearing, lancinating, and burning, and are often considered to be of a rheumatic nature. The paræsthesiæ are described as sensations of pricking or numbness, and are especially localized in the finger tips; here the patients have the feeling of "going to sleep" and numbness, so that they can no longer feel a needle, pick up a small coin, etc. Occasionally pains and paræsthesiæ are combined. Very often also there are abnormal sensations of heat and cold in the tips of the fingers.

The arms and the trunk, as well as the hands, may be the seat of subjective disturbances. In the legs, on the contrary, these disturbances are very rare. If they occur, they consist chiefly in paræsthesiæ of the soles of the feet.

Trophic changes are observed in the skin, the subcutaneous cellular tissue, and the bones and joints.

In the skin are found hyperæmia or anæmia, dermatitis, erythema, herpes, spontaneous gangrene, atrophy or hypertrophy, pemphigus vesicles, hyperidrosis or anidrosis, ulcerations and rhagades. There are also changes in the nails—brittleness, deformities, and distinct longitudinal markings. Panaritium and phlegmon occur in the subcutaneous cellular tissue.

The implication of the bones and joints in the disturbances of nu-

The symptoms which have just been described in detail constitute the type of the disease, *i.e.*, the form in which the process begins in the cervical cord and advances either upwards or downwards (cervical type).

Besides this form, however, there are others, the various anomalies in the course of which present a widely varying picture, so that only the principal forms can be described.

And, first, it may happen that, as has already been mentioned, the affection may develop chiefly in the dorsal or lumbar cord, in which case atrophies and disturbances of sensibility first appear in the lower extremities, and the arms may occasionally escape entirely.

Further, there are cases in which the disturbances are almost exclusively motor, sensory symptoms in the form of pain, paræsthesia, or other subjectively demonstrable abnormalities, appearing quite late in the disease or not at all. If to these are added early contractures of the legs with increase of the patellar reflex, a picture is presented which strongly suggests amyotrophic lateral sclerosis or the complexus of spastic symptoms. We shall return to this subject again when speaking of the differential diagnosis.

The above-mentioned humeroscapular type of the atrophy may be so prominent as to simulate the juvenile form of progressive muscular dystrophy, especially if the sensory manifestations are but slight.

If, on the other hand, the sensory phenomena predominate greatly, the motor sphere being implicated slightly and at a late period, there may be produced an assemblage of symptoms much like hysteria, particularly if, as not rarely happens, the posterior roots and the posterior columns are attacked unilaterally, so that a hemianæsthesia can be demonstrated.

A quite distinct variety of syringomyelia is that form which manifests itself almost solely by trophic disturbances. In these cases, besides the more or less well-marked muscular atrophy, there are found on the hands chaps of the skin in great numbers, painless panaritia, and fissures and thickening of the finger joints. Objective testing of the sensibility reveals also the characteristic partial paralysis of sensation. Cases of this kind were described by Morvan in 1883, and were regarded as constituting a disease *sui generis*, which Morvan himself designated as "parésie analgésique à panaries des extrémités supérieures," or as "parésoanalgésie des extrémités supérieures." But it is now known that the so-called Morvan's disease depends anatomically upon the same process as does syringomyelia, and therefore should not be separated from it. A similar group of symptoms occurs only in lepra; if this disease can be certainly ex-

cluded, a spinal gliosis will always prove to be the anatomical basis of the affection.

Finally, it may happen that the gliosis and the formation of cavities are confined exclusively to the posterior columns. Then, of course, the clinical picture may agree completely with that of tabes dorsalis. It has, moreover, been proved by pathological investigation that syringomyelia may be combined with genuine tabes.

Diagnosis.

These considerations lead us naturally to speak of the differential diagnosis which in syringomyelia covers a wide field, and in many cases is attended with great difficulties.

Atrophies of the hand muscles in connection with spastic phenomena in the legs are, excepting syringomyelia, found especially in amyotrophic lateral sclerosis. But this disease, at least in all typical cases—and others need not now be considered—is dependent upon an anatomical process which is confined to the motor apparatus, *i.e.*, the ganglion cells of the anterior cornua and the corresponding peripheral nerves, never attacking the sensory paths. If, however, a few atypical cases of amyotrophic lateral sclerosis occur in which disturbances of sensibility are present, the latter never extend so widely distributed over considerable portions of the body as in syringomyelia; and, above all, partial paralysis of sensation is never found. The possibility that the medulla oblongata may become implicated is common to both diseases. But the bulbar symptoms in amyotrophic lateral sclerosis, in correspondence with the symmetrical extension of that process, are almost invariably bilateral and symmetrical; in syringomyelia, however, as has already been emphasized, they are unsymmetrical and often unilateral. It must be admitted that those cases of syringomyelia in which the sensory symptoms are very late in appearing, so that at first only atrophy of the muscles of the hand and spastic phenomena in the lower extremities are present, may oppose insuperable obstacles to the differential diagnosis from amyotrophic lateral sclerosis.

Spinal progressive muscular atrophy also begins with atrophy of the hand muscles. But in this affection there are no sensory disturbances and no spastic symptoms of the lower extremities. Moreover, the course of the disease is somewhat more rapid; and, finally, it should not be forgotten that, in comparison with syringomyelia, spinal progressive muscular atrophy is a very rare disease.

The humeroscapular type of syringomyelia may be confounded with the juvenile form of progressive muscular dystrophy. But

in this affection also there are no sensory disturbances, and the changes of electrical excitability which may occur in syringomyelia are wanting.

Caries of the cervical spine may lead to atrophy in the hands, to disturbances of sensibility, and to oculopupillary symptoms; but, at least in the more advanced stages, the presence of vertebral disease may be recognized, or tuberculous affections of other organs may be detected.

Affections of the brachial plexus may simulate syringomyelia. In the so-called paralyses of the upper plexus, known as the Duchenne-Erb paralyses, only the muscles about the shoulder and in the arm (and in the forearm only the supinator) are affected, a localization which would not be found in syringomyelia. On the other hand, paralysis of the lower plexus, Klumpke's paralysis, affects especially the small hand muscles and produces also oculopupillary phenomena. The doubt will be decided by the fact that these plexus paralyses are produced almost solely from traumatism, and also that the disturbances of sensibility which follow them affect all kinds of sensation, instead of being partial, as in syringomyelia.

Multiple neuritis may also be mistaken for syringomyelia. In this also there are atrophic paralyses; in fact, partial paralysis of sensation is not very rare in neuritis. Yet in syringomyelia the severe pain of neuritis and the extreme sensitiveness of the nerve trunks to pressure are absent; and finally, the partial paralyses of sensation do not affect whole segments of the body, as in syringomyelia, but are rather confined to the course of single nerves.

Pachymeningitis cervicalis hypertrophica, besides atrophic paralyses of the hand muscles, presents also pain, trophic disturbances, and disturbances of sensibility, occasionally also even spastic phenomena in the legs. The distinction from syringomyelia may therefore occasionally prove very difficult. It is to be made by the facts that the whole course of pachymeningitis cervicalis hypertrophica is much more rapid, that the disturbances of sensibility almost always affect all kinds of sensation, and that pain is the most conspicuous feature of the disease.

Hæmatomyelia is also more frequently located in the cervical cord, and from this part extends longitudinally through the cord. If it attains some size it may therefore simulate syringomyelia. Decisive for the diagnosis of hæmatomyelia is the proof of a traumatism as the cause, as well as the rapid development of all symptoms, followed by their slow retrogression in consequence of the gradual absorption of the hemorrhage; while syringomyelia also runs a slow course, but is progressive.

That multiple sclerosis may present a similar picture to that of syringomyelia is evident. There are found in it the complexus of spastic symptoms, nystagmus, muscular atrophy, and occasionally also disturbances of sensibility. But very rarely in multiple sclerosis are there so extensive muscular atrophies as in syringomyelia, and, above all, the reaction of degeneration which may be found in syringomyelia is absent here. Moreover, the disturbances of sensibility which occur in multiple sclerosis never possess the character of the partial paralyses of sensation; and finally, trophic affections of the bones and joints, such as are found in syringomyelia, are great rarities in multiple sclerosis. However, it is admitted that multiple sclerosis cannot be excluded with certainty in any diffuse disease of the central nervous system.

The relation of tabes dorsalis to syringomyelia has already been noticed. There are, in fact, cases in which the two diseases cannot be distinguished from each other, especially because of the trophic disturbances which are so extensive in tabes as well as in syringomyelia. As points of difference may be mentioned the facts that in tabes the process generally begins in the legs, in syringomyelia in the upper extremities; that while in tabes atrophy of the muscles of the hands may develop as a consequence of secondary peripheral neuritis, yet this occurs only in the last stages of the disease, but in syringomyelia it constitutes the beginning; finally, that in the large majority of cases of tabes a previous syphilis plays an etiological rôle, while, as was remarked at the beginning, the latter almost never has such a significance in syringomyelia.

The diagnosis between syringomyelia and leprosy may occasionally be difficult. Lepra anæsthetica, in which form alone the question could of course arise, also causes muscular atrophy, especially in the hands, and trophic and sensory disturbances, the sense of temperature especially suffering. On the other hand, there are found in leprosy the characteristic leprous skin affections—nodes with ulcerations, white anæsthetic cicatrices, and in the secretion of the ulcerations the lepra bacillus may be demonstrated, which, of course, establishes the diagnosis beyond doubt. In leprosy the nerves are very sensitive to pressure and are to be felt as painful, thickened cords, which never occurs in syringomyelia. The disturbances of sensibility in leprosy affect almost solely the hands and feet, and in them are developed by preference in the most peripheral portions. They never correspond as in syringomyelia to the individual segments of the spinal cord. Spastic symptoms in the legs are rarities in leprosy. And finally, the fact that the patient in question comes from a leprous region or has

recently dwelt in such a locality, is of importance for the diagnosis of leprosy.

Treatment.

Since we cannot in any way modify the disease process in the spinal cord, it is evident that there can be no hope of a successful treatment of syringomyelia. There remain only, on the one hand, palliative treatment, *i.e.*, securing a quiet life for the patient and, if necessary, treatment of the trophic affections; on the other, suggestive or hope-inspiring treatment—hydrotherapy, baths, massage, and inunctions.

SPASTIC SPINAL PARALYSIS.

Anatomically, the so-called spastic spinal paralysis, also known as lateral sclerosis, consists in a degeneration of the lateral pyramidal tracts. This is found with relative frequency at autopsies. Whether, however, it is proper to regard it as a primary disease, is a still undecided question. There is no doubt that lateral sclerosis occurs more frequently in the train of other diseases of the spinal cord and brain than as a perfectly independent affection without any other changes in the central nervous system. A few autopsies do indeed prove the fact of the occurrence of primary lateral sclerosis, but they must be considered rare exceptions in comparison with the much more numerous cases in which the affection simply constitutes a symptom of the primary disease. Since, therefore, primary lateral sclerosis must be regarded as a rarity, it is better to designate the symptoms which are produced in general by the degeneration of the lateral columns as the spastic symptom complexus. This complexus presents a well-marked clinical picture, which is found in the course of various diseases of the nervous system, to be described in detail further on.

Symptoms.

The disease always begins in the lower extremities with the feeling of tension and stiffness. The patients describe their condition by the expression, "the tendons are getting too short." The once perfectly easy movements of the legs and feet become difficult, the gait is noticeably affected, and all exercise of the lower extremities which involves effort—such as dancing, skating, mountain-climbing, etc.—is either greatly impeded or rendered impossible. Besides the tension, fatigue is soon felt in the legs after very slight exertion. Involuntary twitchings of the legs are very common in this initial stage. The pathological condition is not always equally developed in both

legs. One leg alone may be attacked, the other showing the same symptoms only at a much later period.

In the further course of the disease genuine contractures develop in addition to the weakness and stiffness of the muscles. These affect the adductors and extensors, especially in the thigh and in the leg, the flexors of the feet, causing abnormalities in the position of the extremities. Upon examination of the patients the following condition is discovered: The active mobility of the leg is nowhere abolished, but is much impeded, because in every movement the excursion of the joint cannot be fully carried out on account of the tension of the muscles. If passive movements are performed—if, for example, one attempts to move the thigh at the hip-joint, a certain force is necessary to overcome the tension of the adductors which fix the thigh. In severer grades of the affection such movement is actually impossible. It can be demonstrated that the muscles are in part distinctly contractured, especially the adductors of the thigh and the flexors of the feet. In consequence of the contracture of the adductors the legs cannot be sufficiently separated, and the knees strike against one another.

The affection is most manifest in the gait, which is called the "spastic gait." Since the legs are kept flexed at the knee-joint because of the contracture of the flexors of the back of the thigh, the complete extension of the knee necessary for normal walking cannot be effected. The foot, on account of the contracture of its flexors, is planted only by the toes, never with the whole sole—it gives the impression that the patients are glued to the floor. They drag their feet forward painfully by very small steps, seeking to compensate for the insufficient mobility of the thigh at the hip-joint by thrusting forward the whole pelvis. Their shoes are always worn through first or even exclusively at the tip, never in the middle of the sole.

The second peculiarity of the spastic complexus is the enormous increase of the tendon reflexes. A quite gentle touch upon the tendon of the quadriceps suffices to excite the patellar reflex. An extension of the leg is then produced, which may pass into a tonic contraction of the quadriceps, lasting for several seconds, so that we speak of a patellar tonus. Very often the patellar reflex is excited in the intensity just described by simply seating the patients upon a table so that their legs hang free. The pull upon the quadriceps tendon thus induced is sufficient to cause the reflex. Attention has recently been directed to another phenomenon connected with the quadriceps tendon, which is dependent upon the increased reflex excitability. If the patella is pulled forcibly downwards, a strong clonic contraction of the whole quadriceps is produced. In the severer

forms of the spastic complexus the so-called dorsal or foot clonus is always present. If in passive extension the foot is forced strongly upwards, in consequence of the resulting tension of the tendo Achilles, there occurs a vigorous contraction of the calf muscles, which produces a quivering of the whole calf, and which may even be communicated to the entire extremity.

Otherwise there are no nerve disturbances in the legs. The muscles do not atrophy, the electrical reaction remains unchanged, the sensibility continues to be normal, trophic changes do not occur, and the functions of the bladder and of the rectum are never disturbed. The contractures and the increased tendon reflexes are the characteristic features of the disease.

Occasionally the same process is found in the arms, and here, too, there are contractures and spasms of various muscles, combined with an increase of the tendon reflexes. The triceps tendon reflex is much exaggerated, and the periosteal reflex is also increased. But the spastic symptom complex is very rare in the arms.

Etiology.

The cause of these phenomena, as has already been stated, is to be sought in the degeneration of the lateral pyramidal tracts. It is probable that centrifugal fibres lie in these tracts, which are to be regarded as reflex inhibitory in function. Lesions of these fibres would naturally produce an increase of the tendon reflexes. The cause of the contractures, however, has as yet not been demonstrated.

The spastic complex affecting the legs is found in three diseases of the nervous system, namely, hydrocephalus, encephalitis, progressive paralysis. Of diseases of the spinal cord are especially to be mentioned chronic myelitis of the cervical and dorsal portions, syringomyelia, hydromyelia, insular sclerosis, amyotrophic lateral sclerosis, and, finally, the combined system diseases as soon as they have involved the lateral tracts.

Hysteria may also cause the complex of spastic symptoms, and spastic phenomena may occur in lathyrism and in pellagra.

The diagnostic problem, therefore, when the complexus of spastic symptoms is present, is to demonstrate the existence of one of the diseases just enumerated by means of the other symptoms, and thus to prove the dependence of the complex upon it. And it should not be forgotten that many of the diseases mentioned often reveal themselves for years only by the spastic symptom complex, until finally a symptom which does not belong to it appears and makes the primary disease known. This is especially true of insular scle-

rosis. We must not therefore conclude with absolute certainty from the absence of other symptoms than those of the spastic symptom complex that a case is one of unmixed primary spastic spinal paralysis, for death may finally occur from intercurrent disease before the primary affection has had time to manifest itself.

Prognosis and Treatment.

The prognosis and treatment vary with the original disease. Nothing is to be expected from treatment, except in case of syphilis. Then an energetic treatment by inunctions should be instituted, for treatment, if too late, is useless. If atrophy or cicatricial tissue have developed in the intraspinal tissues, mercury is no longer of benefit. Aside from the use of mercury, thermal waters and saline baths are the most important remedial measures in the unsatisfactory treatment of these chronic diseases of the spinal cord.

AMYOTROPHIC LATERAL SCLEROSIS.

Amyotrophic lateral sclerosis—better named myoatrophic lateral sclerosis—as its name implies, consists in an affection of the ganglion cells of the gray anterior cornua—atrophy—combined with spastic paralyses—lateral sclerosis.

Pathological Anatomy.

Corresponding to these affections, there is found a degeneration of the anterior and lateral pyramidal tracts, which may be followed from the lower lumbar to the cervical cord, but very often from the latter region invades also the higher motor paths, *i.e.*, the motor paths in the pons, the crura cerebri, the internal capsule, and even the central convolutions. Simultaneously with this process there develops atrophy of the ganglion cells of the anterior cornua, which begins generally in the cervical cord and advances downwards. Finally, the atrophy also attacks the nerve nuclei in the medulla oblongata, so that the nuclei of the facial, hypoglossus, trigeminus, and vagus nerves may become affected. The peripheral motor nerves which proceed from the ganglion cells of the anterior cornua or from bulbar nuclei manifest degeneration also, although in a lesser degree. The muscles which depend for their nutrition upon the diseased ganglion cells are, of course, attacked by degenerative atrophy. The remaining portions of the spinal cord, especially the other tracts of white matter, remain perfectly normal.

Etiology.

Very little is known with regard to the etiology. Exposure to cold, overexertion, psychical excitement, and trauma have been thought to be causative, but their influence cannot be positively proved. The disease begins between the twenty-fifth and forty-fifth years. Men are attacked with decidedly greater frequency than women.

Symptoms.

The beginnings of the disease consist always in a weakness of the upper extremities, especially of the hand muscles, and of a stiffness in the legs, which easily become tired. There is also unsteadiness of gait. Soon the first atrophies appear in the hands. These, as a rule, attack first the ball of the thumb and the hypothenar eminence, then the interossei. A corresponding loss of function is produced, and through the preponderance of the antagonists the hand soon becomes "clawed" (*main en griffe*). The atrophy then advances with comparative rapidity, and attacks the extensors of the forearm, the deltoid, and the triceps. The muscles very frequently exhibit fibrillary twitchings. Electrically there is either no change or a quantitative lessening of excitability; in certain muscle fibres, especially of the hand muscles, a distinct reaction of degeneration may be detected.

Besides these atrophies, contractures also soon appear; the arms are in the position of adduction, the forearms are flexed, the hands are either pronated or strongly flexed. Moreover, the power of the whole arm is more diminished than appears to correspond with the condition of the muscles first atrophied. In muscles in which atrophy can hardly be demonstrated, there is already great loss of power.

At an early period a considerable increase in the tendon reflexes in the arms is manifested; the periosteal as well as the tendinous reflexes are extremely vigorous. A blow upon the tendons of the supinator longus or of the triceps, as well as upon the radius, calls forth active muscular contractions.

Sensibility remains quite normal in the great majority of cases; in very few, paræsthesiæ and slight pains in the hands and arms are described. Trophic changes do not occur.

In the lower extremities rigidity of the legs is from the first the most conspicuous feature, atrophy in this region being comparatively quite unimportant. The muscles of the legs show great motor weakness and loss of force. Active and passive spasms, often severe, are also frequently developed. The tendon reflexes are extremely exag-

gerated; foot clonus can be elicited in the majority of cases. The spasms produce a spastic-paretic gait, the patients touching the ground almost solely with the tips of the toes and dragging themselves along with small steps. The entire complexus of spastic symptoms becomes thus developed. In the legs there are never disturbances of sensibility. The bladder, the rectum, and the sexual functions are also never affected.

After a duration of years the process finally invades the medulla oblongata, and bulbar symptoms are developed. These manifest themselves as atrophy of the lips, in consequence of the implication of the nucleus of the facial, and as atrophy of the tongue from lesion of the hypoglossus—both atrophies produce dysarthria. If the vagus is affected, disturbances of deglutition, abnormalities of the heart's action, and occasionally also serious disturbances of respiration are produced. If the degeneration attacks the nucleus of the trigeminus, paresis of the masticatory muscles occurs. The sensory fibres of the medulla nerves likewise are never implicated. The reflexes in the regions supplied by the nerves from the medulla are much increased; there is a particularly great increase in the masseter reflex.

The affection lasts from four to six years. Of course, the prognosis is always absolutely unfavorable, and therapy is powerless. Death generally takes place from paralysis of respiration.

Diagnosis.

This is not difficult, if it be remembered that amyotrophic lateral sclerosis attacks only the motor apparatus, and that it consists in the singular combination of atrophy of the arms with spasms of the legs. Every other disease which presents this complex of symptoms in connection with disturbances of sensibility, can therefore be excluded. Syringomyelia, for example, might be suspected, for this disease also first causes atrophy of the hands and then spasms of the legs, if it were not that there are always marked disturbances of subjective and objective sensibility. Only when the posterior cornua escape in syringomyelia, which sometimes happens, is there a clinical picture which much resembles that of amyotrophic lateral sclerosis.

In chronic myelitis atrophies of the arms and spasms in the legs are sometimes met with likewise; but at the same time there are always more or less pronounced disturbances of sensibility, and especially of the bladder and rectum.

Multiple sclerosis, besides the symptoms characteristic of amyotrophic lateral sclerosis, which may also occur in it, presents scan-

ning speech, intention tremor, and atrophy of the optic nerve; occasionally it cannot be excluded with absolute certainty.

The spinal form of progressive muscular atrophy, which always begins in the muscles of the hand, is attended neither by disturbances of sensibility nor by spasms of the legs.

Pachymeningitis cervicalis hypertrophica may also be attended by paresis and atrophy in the arms and spasms in the legs, but has a more rapid course; pain and disturbances of sensibility are also present.

Peripheral polyneuritis may occasionally manifest the complex of symptoms belonging to amyotrophic lateral sclerosis in the arms, but will always show, besides these, disturbances of sensibility, and there will be no increase of the tendon reflexes.

Finally, the bulbar form of amyotrophic lateral sclerosis may be confounded with other diseases of the medulla oblongata. To be considered are the acute bulbar paralyses from hemorrhage or softening, which are distinguished by their suddenness of onset and chronic progressive bulbar paralysis. The latter is, of course, quite identical with the bulbar form of amyotrophic lateral sclerosis, and is distinguished from the purely spinal form of the disease only by the absence of atrophy in the hands. But the two diseases may be combined with one another.

Treatment.

Amyotrophic lateral sclerosis is incapable of being benefited by treatment. In the absence of anything better, electrotherapy is employed.

PROGRESSIVE SPINAL MUSCULAR ATROPHY.

The anatomical basis of this affection is a lesion of the motor apparatus from the gray anterior cornua to the muscles, the motor ganglion cells, the anterior nerve roots, the peripheral motor nerve, and, finally, the muscles also falling a prey to degenerative atrophy.

Pathological Anatomy.

The details of the anatomical picture are as follows: the anterior cornua are diminished in size; the ganglion cells of the cornua have wholly or in part disappeared; those cells which are still present are atrophied, *i.e.*, the cell itself has become diminished; the nucleus is contracted, and the processes are wasted. The anterior roots are granular and have undergone fatty degeneration. The motor fibres

for the most part exhibit only a partial degeneration; besides fibres which are still perfectly normal are found others with fatty or pigmentary degeneration, as well as with interstitial proliferation of connective tissue. The atrophic muscles show in part only simple atrophy, *i.e.*, considerable diminution in thickness of the fibres with preservation of their transverse striation; but for the most part there is degenerative atrophy—splitting of the fibres, increase in the number of the muscle nuclei, proliferation of the interstitial connective tissue, and fatty degeneration of the muscle substance.

In this disease the white matter of the spinal cord remains quite intact. The few cases in which besides the changes already mentioned a degeneration of the lateral tracts was also found, must be regarded as atypical, and in view of their great rarity need not be considered.

Etiology.

Practically nothing is known with regard to the etiology of spinal muscular atrophy. Sometimes over-fatigue of the hands, as in washing, threshing, etc., may seem to play a part in the causation; but by no means always. Heredity also appears to be of little influence. Men are more frequently attacked than women.

Symptoms.

The disease begins between the twentieth and fiftieth year, with slowly increasing weakness of the upper extremities, especially of the hands. It becomes difficult for the patient to perform delicate movements with them. But pain or other disturbance of sensibility is entirely absent. After a time atrophy manifests itself in the hands, generally first attacking the adductor and opponens pollicis, but soon involving the other muscles of the hand, the whole thenar, the hypothenar, and the interossei. Hence arise the familiar sinking of the interosseous spaces, the wasting of the ball of the thumb and of the little finger, and finally, through the preponderance of the flexors and extensors of the forearm, the *main en griffe*. As the atrophy extends it overleaps usually the muscles of the forearm, to next attack the shoulder muscles, especially the posterior and upper third of the deltoid. Then the biceps is affected, after that the muscles of the forearm, the extensors at an earlier period than the flexors. The triceps is generally spared for a very long time. The muscles of the trunk are next implicated in the atrophy; first the trapezius, next the pectoralis, the rhomboideus, and the latissimus dorsi, the muscles of the throat and neck generally escaping. The atrophy very rarely oc-

curs in the lower extremities. If it does appear here it is only in the last stages of the disease.

The course of the atrophy just described, which begins in the small muscles of the hand and advances gradually in a centripetal direction—known as the Duchenne-Aran type—is the most common but not the only form of the disease. Thus it may happen that the atrophy begins in the shoulder muscles and only subsequently attacks the muscles of the hand.

All the atrophies in this disease are well marked. Since the subcutaneous connective tissue and the fat participate in the atrophy, the contours of the bones are everywhere sharply prominent. The atrophied muscles show almost without exception strong fibrillary contractions, which may even be manifested in muscles which have not been visibly affected by the disease. There are various changes in the electrical excitability, according to the condition of the affected muscle or nerve. If a considerable number of healthy nerve and muscle fibres are still present, there is only a more or less pronounced quantitative diminution of the excitability, which, as the degeneration progressively increases, may finally be entirely lost. With the increase of the degeneration and atrophy of the ganglion cells of the anterior cornua, their trophic influence proportionately diminishes and the reaction of degeneration becomes manifest in the muscles. This reaction is, of course, only demonstrable in some of the fibres, because of the slow progress of the anatomical changes. It will therefore be necessary to examine each bundle of fibres individually, in order to detect abnormal slowness of contraction. By some authors an increase in muscular excitability has been described as occurring in muscles which are not yet visibly atrophied, this increase thus leading to the formation of a bad prognosis. This condition, however, seems not to be constant.

The tendon reflexes of the arms are, of course, completely abolished. This is true also of the legs as soon as the disease attacks them. Disturbances of the sensibility are completely absent, nor are there any subjective abnormalities of sensation in the form of pain, paræsthesia, etc.

But, on the other hand, slight vasomotor and trophic disturbances may occasionally develop; above all, a certain coldness and cyanosis of the skin over the atrophied muscles of the hand; also cracks and thickenings of the skin, and brittleness and splitting of the nails. The bladder, the rectum, and the genital functions always remain intact.

Occasionally in the further course of the disease the anatomical changes attack the nerve nuclei of the medulla oblongata with their

peripheral nerves, and bulbar affections develop in the form of atrophy of the tongue, with consequent difficulties of speech, disturbances of mastication and of respiration, partial paralysis of the facial nerve, etc.

The course of the disease is always very chronic, extending often over decades. Remissions hardly ever occur; the disease advances irresistibly, though slowly. Death occurs either from intercurrent disease or from paralysis of respiration, in consequence of the implication in the atrophic process of the respiratory muscles or from lesions of their bulbar centres.

Diagnosis.

This is very easy in the pronounced cases, if the significance of the facts that the disease begins in the hands and that the sensibility is not impaired be appreciated.

Amyotrophic lateral sclerosis also produces atrophy of the muscles of the hand, the sensibility remaining normal, but is always accompanied by spastic phenomena in the legs and increase of the tendon reflexes in the arms.

Pachymeningitis cervicalis hypertrophica, which may also cause atrophy of the hand muscles, is always attended by phenomena of sensory irritation in the arms, and produces especially objective disturbances of sensibility.

Caries of the cervical spine causes not only atrophy, but also objectively demonstrable changes of sensibility in the hands. Moreover, at least in its more advanced stages, sensitiveness of the cervical vertebræ on pressure or a gibbus in that region can always be detected.

In syringomyelia, the disease which may most resemble spinal progressive muscular atrophy with respect to the degree of the atrophy of the hand muscles, the atrophies are generally unsymmetrically and irregularly developed in the two hands, and extensive subjective and objective disturbances of sensibility are always conspicuous features of the disease.

The distinction from the myopathic forms of progressive muscular atrophy, the so-called progressive muscular dystrophy, will be necessary only in the few exceptional cases in which spinal progressive muscular atrophy begins in the shoulder muscles, precisely like the juvenile form of the dystrophy. Aside from the fact that their further course will differentiate the diseases—the dystrophy does not attack the muscles of the hands—a distinction can be made by the behavior of the atrophic muscles to electricity; in dystrophy there is no reaction of degeneration.

curs in the lower extremities. If it does appear here it is only in the last stages of the disease.

The course of the atrophy just described, which begins in the small muscles of the hand and advances gradually in a centripetal direction—known as the Duchenne-Aran type—is the most common but not the only form of the disease. Thus it may happen that the atrophy begins in the shoulder muscles and only subsequently attacks the muscles of the hand.

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TABES DORSALIS.

BY

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LEIPSIC.

TABES DORSALIS.

Synonyms.—Locomotor ataxia; *Rückenmarksschwindsucht;* Sclerosis of the posterior columns.

That such a well-marked disease as tabes should have been first recognized as a distinct affection only about fifty years ago is not flattering to the medical profession. It is true that many statements of the older physicians are interpreted to apply to tabes, but these expressions are so indefinite that no particular weight is to be given them. It is generally stated that Duchenne was the actual discoverer of tabes. This is not strictly correct, inasmuch as Wunderlich and others had given very good accounts of the disease before him. Nevertheless the admirable description, worthy of a clinical genius, given by Duchenne, who knew nothing of the work of his predecessors, deserves the highest praise. From Duchenne's time until today there has flowed an ever-widening stream of articles upon tabes. The essays devoted to this subject up to the present time number about two thousand, and every year enlarges and deepens our knowledge of the disease. It is impossible to give here an adequate review of the literature of the subject. Those who are interested in it are referred to special works.* If the fact that the sharply marked peculiarities of tabes were long unrecognized is not complimentary to the perspicacity of our ancestors; on the other hand, the fact that year by year tabes is becoming more and more common throws an unfavorable light upon the morals of modern life. We know now that tabes is metasyphilis, *i.e.*, a sequel of syphilis. Tabes becomes more frequent in just the degree that syphilis extends. We should not, therefore, regard ourselves as extraordinarily clever, for it is now easier for us to study tabes than it was for the earlier physicians.

Like tabes, general paralysis of the insane was late in being recognized but becomes more frequent year by year. The two diseases, in my opinion, are essentially one—metasyphilis of the nervous system. Their localization alone differs, that is, if the brain is especially diseased we speak of general paralysis; in case the centripetal nerve

*I have regularly reported the articles on tabes in Schmidt's Jahrbücher der gesammten Medicin since 1879. In these reports all of the important modern contributions are reviewed.

fibres are particularly affected the term tabes is used. The historical development of our knowledge, however, and the usage of practical medicine separate tabes from general paralysis. This usage, which classifies the former with diseases of the spinal cord and the latter with mental diseases, is followed in this work.

Etiology.

The ignorance as to the causes of tabes which at first prevailed led investigators to take into consideration all the conditions which could possibly have a share in the origination of the disease. It is therefore surprising that the connection between syphilis and tabes was pointed out only at a relatively late period and that this etiology found at first little acceptance.

Duchenne was of the opinion that syphilis often seemed to be "the only rational" cause of locomotor ataxia, but that since the symptoms were the same as in the other cases and since antisyphilitic treatment was of no benefit this supposed etiological relationship did not exist. Eisenmann and E. Schulze, indeed, pointed out the importance etiologically of syphilis, but their data were insufficient. It was not until 1876 that A. Fournier expressed emphatically the opinion that syphilis is one of the most frequent causes of tabes. Vulpian sympathized with his views but believed that syphilis merely predisposed to tabes. In the year 1879 W. Erb first supported Fournier's view; of 30 patients with tabes Fournier found previous syphilis in 24, Erb found 27 out of 44 cases with a syphilitic history. In the same year Erb reported the results of an examination, as a control-experiment, of 85 men over twenty-five years of age, among whom only 14 were found to have had syphilis. Since that time Erb has been led, by a constantly increasing accumulation of statistics, to support Fournier's views most strongly. After his first publication statistics were collected from all quarters, and some of them were of a very unsatisfactory nature. Old histories of cases were examined, the cases of tabes in which antecedent syphilis was noted were collected, and the report was made that only 20 or 30 per cent. were found to be syphilitic.

The Berlin neurologists, Westphal, Remak, Bernhardt, and especially Leyden, attacked the new doctrine with especial energy. It found opponents in France also. Julliard and Charcot thought that syphilis of the spinal cord had an appearance differing from that of tabes. Charvot and his school declared that a "nervous heredity" was without question the cause of tabes. In subsequent publications expressions of opinions for and against the

theory alternated with each other. L. Meyer, of Berlin, found not one case with previous syphilis among 19 cases of tabes in women. A strong supporter of Fournier's view appeared in W. R. Gowers, who found 70 per cent. of cases of tabes to be syphilitic (1881). In 1881 Erb published for the first time statistics dealing with large numbers of cases. He found of 100 patients with tabes 88 per cent., among 400 patients with other diseases only 23 per cent. to have had antecedent syphilis. The question was discussed at the international congress in London where Erb vigorously defended his views.

In 1882 Fournier again declared that in the vast majority of cases tabes was of syphilitic origin. Since 1875 in 117 cases of tabes he had found 91.45 per cent. to have had syphilis. He emphasized the facts that tabes begins almost always in the tertiary stage (in 85 cases out of 89 after the third year) and that the syphilis had generally been mild. (Of 84 cases, in none had the disease been very severe, 10 had had syphilis of medium severity, and 61 benign syphilis.) In 1883 Erb reported 200 cases of tabes; 9 of these were without syphilitic infection. Of 1,200 cases of other diseases he found 22.75 per cent. to be infected. In 1884 Oppenheim found out of 100 cases of tabes 59 without syphilitic infection. Bernhardt in four successive groups of statistics computed that 40, 60, 57.6, and 83 per cent. of cases were syphilitic. This is a good example of the way in which the percentage grows with increasing care in the investigation of the history of the patients.

Since 1883 American neurologists have taken part in the controversy, among whom may be mentioned Birdsall, L. Weber, and Seguin. The last-named found 72.22 per cent. of tabes cases syphilitic. In 1884 I was able to declare from the statistics of Fournier and Erb, as well as from Erb's study of series of cases of other diseases, that the connection between syphilis and tabes is as well proven as anything could be which is not susceptible of exact demonstration, that negative do not disprove positive statistics, that there are numerous facts besides those derived from statistics which lead us to infer the connection, that the objections of opponents only show that tabes is not tertiary syphilis, that these objections lose their weight if tabes is regarded as a sequel of syphilis and that with our present knowledge it is proper to view syphilis as the *conditio sine qua non* or as the primary cause in all cases of tabes. Strümpell gave his adhesion to the view that tabes is a sequel of syphilis, having found 90 per cent. of his tabes patients to be syphilitic. He thought that a toxin originates in the body of the syphilitic, and that this toxin produces tabes. But neither he nor others ventured to deny the existence of "non-syphilitic tabes."

In 1885 another important work by Fournier appeared. Of 146 new cases of tabes there were only 9 in which Fournier thought that syphilis should be excluded, and these statistics are the more valuable because all the important details are given for each case. In the two series of statistics of Fournier 93 per cent. of the cases were syphilitic. Further study showed that the frequency of occurrence of syphilis was paralleled by that of tabes in different classes and peoples. Thus Minor found that the latter was as rare as the former among the Jews, and that both were comparatively frequent among the Russians. Several authors pointed out the rarity of both diseases among the clergy and among Quakers. The correctness of Fournier's doctrine came to be more and more widely accepted. Oppenheim admitted it without reserve in 1889, as did also F. Raymond. And the necessity of holding tabes to be always the result of syphilis also became recognized. D. Drummond and P. Marie expressed that view and Strümpell was inclined to accept it. Erb continued unwearied in the collection of material. In 1896 he had at his disposal the histories of 700 cases of tabes, of which 90.35 per cent. were syphilitic. Among his last 200 cases there were only 4 in which infection was not probable.*

The following may be regarded as now certainly proven: In the great majority of cases of tabes it is possible to demonstrate the previous existence of syphilis. Tabetic patients in whom such infection is improbable are extremely rare. Before we proceed to draw further conclusions from this let us examine more closely the conditions under which tabes appear.

Age.—Tabes is extremely rare in children. If the literature of the subject is explored we may succeed in collecting perhaps twelve or fifteen cases in which tabes began before the fifteenth year. But in almost all of these cases, it was possible to demonstrate hereditary syphilis, as was the case in some of Fournier's patients, in two of Gowers', in three of B. Remak's, in one of Strümpell's, etc. Almost as rare is tabes in old age. O. Berger, however, reported a tabetic patient, seventy-four years old, who had been ill for two years. He was infected with syphilis in his seventieth year. In about two-thirds of the cases tabes begins between the thirtieth and fortieth years, rarely before the twenty-fifth, and rarely after the fiftieth year. All authorities are pretty well agreed in this.

Sex.—Tabes at the usual age is much more frequent in men than in women. But this difference does not exist in tabes (and general paralysis) of the young, in which the female sex is affected with a

* A detailed exposition of the history of the question of the syphilitic etiology of tabes is to be found in my "Neurologische Beiträge," Heft iii.

slightly greater frequency. Statements as to the numerical ratio of the frequency of the disease in the two sexes vary greatly. Erb had only 32 women among 600 cases of tabes, Fournier only 2 women in 112 cases. Some German authors find a ratio of 1:8; in Berlin the ratio was found to be 1:4.4. Such differences depend manifestly in part upon the kind of clientèle, in part upon the composition of the population. All authors state that tabes is much more rare among the women of the well-to-do classes than among the poor. Among 40 female cases of tabes I found only 1 woman belonging to the upper classes. I have personally examined about 50 tabetic women. In the great majority previous syphilis was certain or highly probable; in not a single case was it improbable. The average age of the patients was thirty-five years; the average interval between the syphilis and the beginning of tabes eight years. Of 32 tabetic female patients of Erb's 14 were certainly, 12 very probably infected; in only 2 was there no evidence of infection. Recently Erb has reported 9 additional cases of which 6 were certainly, 2 almost certainly, and 1 very probably infected with syphilis.

Country and Class.—In the so-called civilized countries tabes probably occurs everywhere with about equal frequency, and its relations to age, sex, and class are everywhere the same. That it is much more rare among barbarous than among civilized peoples seems also to be certain. It is here alone that the frequency of tabes and that of syphilis do not appear to coincide. Reports of peoples among whom syphilis is said to be widespread, but tabes very rare, are frequent. It is true that the statements as to the frequency of syphilis often rest upon very insufficient foundation, but the fact may be admitted to be in general correct. If this be the case, we must suppose that certain secondary conditions are necessary if tabes is to follow syphilis, and these must be left for the future to make clear. At all events, this lack of parallelism between the two affections does not obtain everywhere, for among the Arabs of North Africa, for example, more careful investigation has shown that the occurrence of tabes and of general paralysis keeps pace with the extension of syphilis.

In civilized lands there is a contrast between city and country. In the country tabes is much more rare than in the city, and the larger the city the more frequent the disease. It is more correct, perhaps, to say that tabes increases in proportion to the facilities of communication. The closer the relations between city and country (service in the army, railroads, etc.), the more does tabes extend into the country; and, on the other hand, the more isolated the region or the community, the more rarely is tabes found.

The difference between the classes of a community is very clearly

marked. Although, as has already been remarked, the women of the so-called higher classes are much less frequently affected than those of the lower, in men the reverse is true. In them the majority of tabetic patients are found among the "cultured." The disease is more frequent in private practice than among the poor. It was in private practice that Erb's great number of cases was obtained. Among 550 of his cases there were 324 merchants and manufacturers, 50 officers, 34 lawyers, 24 professors, teachers, etc., and 1 clergyman. The rarity of tabes in ecclesiastics is quite surprising. Bouchard has demonstrated this to be true also of the French clergy with respect to their liability to general paralysis.

We may sum up what has been already said somewhat as follows: Tabes, an extremely chronic disease, begins as a rule at a definite age, within the third and fourth decades, and with few exceptions spares the first two decades and old age. It is much more frequent in men than in women (in great cities four to five times, in general perhaps seven or eight times as frequent). It is the more frequently met with the larger the city. It is very rare in certain classes (especially the clergy), but has a predilection for other classes (merchants, officers, literary men). How are these singular relations to be explained? They point to social conditions, and yet we should hardly expect that the occurrence of a disease like tabes would vary with the sex and position of the patients. We may therefore well inquire if similar relations are met with in other affections. Yes, but really only in two diseases—general paralysis and syphilis. The former appears under precisely the same circumstances as tabes, and the latter differs only in that it makes its appearance at an earlier age—on the average, in the third decade. The explanation is not at first sight easy with regard to tabes and general paralysis; but the influence of social conditions upon the dissemination of syphilis is perfectly clear. Syphilis is acquired at the age when the sexual instinct is most active and its legitimate gratification is often not possible, that is, between the twentieth and thirtieth years. To these cases of adult life, which constitute the vast majority, are to be added, on the one hand, those in which syphilis is present from the beginning, that is to say, is congenital, and on the other, those in which the disease is acquired late in life, after the fortieth year.

It is worthy of note that in the tabes of childhood the difference between the sexes is absent, which is explained by the fact that hereditary syphilis attacks both sexes with about equal frequency. In acquired syphilis the male sex must be preponderatingly affected, since its source is prostitutes, each of whom infects a large number of men, while the latter only exceptionally spread the disease still

farther. In the lower classes sexual intercourse is more facile than in the higher, hence syphilis is readily disseminated among women who are not professional prostitutes. The men of the upper classes gratify their sexual appetite for the most part with the women of the lower classes, marry comparatively late in life, and, if they have become infected, generally take the precaution to postpone marriage for several years. The number of prostitutes and the laxity of morals are greater in the city than in the country and increase with the size of the city. The classes the great majority of whose members are virtuous are spared; those who pride themselves upon being "men of the world" furnish the largest number of victims. Thus all the apparent inconsistencies are at once and completely explained and can only be explained by the relation of tabes to syphilis. What seemed mysterious becomes at once clear if we accept this connection. The chronic disease, tabes, follows in the footsteps of the chronic disease, syphilis, but years always intervene. The interval between syphilis and tabes varies greatly. It may range from two to twenty years, in the majority of cases it varies from five to fifteen years, and its mean duration is from seven to eight years. Everything that has been said of tabes applies with equal force to general paralysis. Whether it be regarded as tabes of the brain or as an independent disease, the conclusions drawn from the study of tabes are equally true of it, each chain of inferences strengthening the other.

We have then in the first place the statistical proof that the great majority of tabetic patients are also syphilitic; secondly, the proof that the peculiarities of the occurrence of tabes can be understood only on the supposition of antecedent syphilis; and thirdly, it remains to be demonstrated that only a slowly acting poison is conceivable as the cause of tabes.

Tabes begins with lancinating pains in the legs and with vesical disturbances, often also with impairment of the sight; there are found at the onset reflex iridoplegia and absence of the knee-jerk; and, as a rule, both sides of the body are affected about equally. From these facts we must conclude that the noxious agent which causes tabes is present in the whole body, for one which acted locally could not affect the head, the bladder, and the legs at the same time, and could not produce approximately symmetrical symptoms. Now a noxious agent cannot affect the entire body unless it circulates in the blood. The cause of tabes must therefore be in the blood. Since, however, certain parts only are affected, but the same parts in all peoples, all classes, and in both sexes, the substance contained in the blood must be able to make a selection. Mechanical conditions in the human body which could explain this selection are not to be found.

The functions of organs cannot be responsible for it, for the tabetic exercises no function which is not also exercised by other men. Consequently we can conceive of but two possibilities, viz., the power of selection exercised by living beings, and chemical affinity—in other words, we have to do either with bacteria or with a purely chemical poison. Anatomical investigations lead to the same result. This also discloses approximately symmetrical changes in different parts of the body, in the optic nerve, in the midbrain, in the processes of the spinal ganglion cells, and in the peripheral nerves. These changes consist in the death of certain nerve fibres and cells, certain groups within the fasciculi which subserve certain definite functions being alone injured. From the simple appearance of the dissected tabetic lesions one can conclude that such changes could be produced only by a poison which circulates in the blood. But what poison except that of syphilis could come into question?

Most authors have not been able to escape the force of all these arguments. They believe, therefore, that tabes is "often" or "as a rule" the result of syphilis, or they say that syphilis is "the most important cause" of tabes. I believe that we should not stop here, that we must regard syphilis as absolutely and always the cause of tabes. According to the most trustworthy statistics ninety per cent. of the cases is accounted for, the question now is with regard to the last ten per cent. The leaders in the controversy, Fournier and Erb, are checked by this last ten per cent, and it is urged against their views that syphilis cannot always be the cause of tabes because in one out of ten cases it is not possible to demonstrate the infection. I should say we are rather to wonder at our success in ninety per cent. in view of the difficulties, the obstinacy with which syphilis is denied, and the frequency of unrecognized or concealed syphilis. Hirschel, of Vienna, has found that in tertiary syphilis, when there was no doubt whatever of the diagnosis, it was not possible to prove infection in more than one-third of the cases. But, aside from statistics, the voice of reason has also a right to be heard. When a disease with marked characteristics is found it surely occurs to no one to suppose that such a disease has now this, now that cause. Is it believed that malaria arises to-day from plasmodia and to-morrow without them, or that tetanus is produced at one time by Nicolaier's bacillus, at another by taking cold? If in ten cases of epidemic cholera the vibrios are found in nine but not in the tenth, it is surely not thought that the tenth case originated, say, from dietetic errors, but the remaining nine from the cholera bacillus. But where is there a disease that is more *sui generis* than tabes, which is unique in the perfect distinctness of its minutest characteristics? And this is the disease

which is said not to have a single source! The longer I reflect upon it the more firmly I believe that tabes never originates without syphilis, and I am convinced that this view will become universal at a not very distant time. Tabes and general paralysis are metasyphilis, or metasyphilitic nerve atrophy, *i.e.*, primary atrophy of nerve tissue, the *conditio sine qua non* of which is syphilis.

From the beginning the opponents of this view have laid stress upon two facts: First that the anatomical changes of tabes do not resemble those recognized elsewhere as syphilitic; second, that mercury and iodine are of no value in tabes. These facts are correct. Tabes is parenchymatous atrophy, not syphilitic neoplastic growth, and antisyphilitic treatment has not the same effect upon it which it has upon the formation of gummata. In so far tabes does not resemble the other manifestations of syphilis. This fact and the relatively late development of tabes after syphilis I recognized in 1884 when I denominated the former a sequel of the latter. The term "metasyphilis" carried with it the same significance. I intended to express nothing by the name as to the mode of the causal connection, with regard to which nothing is as yet known. We can only refer to analogous relationship. As Strümpell has correctly pointed out, tabes stands in the same relation to syphilis as diphtheritic paralysis to diphtheria. The differences are explained by the difference between syphilis and diphtheria. The latter is an acute, the former a chronic infectious disease, and tabes develops at a later period after infection and pursues a more chronic course than diphtheritic paralysis, in correspondence with the chronicity of the original affection. That the tabetic changes are as a rule incurable is explained by the fact that in the parts of the nervous system which are attacked by it no regeneration takes place in any case; the lesions of the spinal cord after smallpox, for example, are also incurable. In so far as the peripheral nerve fibres are primarily affected in tabes, the tabetic symptoms are curable. The most extraordinary feature is the progressive character of tabes. Should it not follow its prototype, syphilis, with respect to its course also? We can, it is true, remove the syphilitic symptoms by treatment, but we are not really able to check the course of syphilis. In the strict sense of the word neither disease is always progressive, for in many cases tabes may cease to advance for the remainder of the patient's life. It is to be noted that tabes also not unfrequently develops in exacerbations as does syphilis. Strümpell's view that, in contrast with gumma, tabes arises not from the syphilis bacteria themselves but from a toxin derived from them has been widely approved. But at present the view is held that the tertiary products also originate not from the simple presence of the bacteria

but from the poison excreted by them. We should therefore be obliged to assume the existence of toxins of various kinds. But all these speculations are in the air, above the realm of facts. This is even more true of Hitzig's hypothesis, which assumes that tabes owes its existence to a peculiar poison which may be present not only in the syphilitic chancre but in the soft chancre as well. I think that the clinician should content himself for the present with the facts, and that he should let the bacteriologist and the chemist do their part towards the solution of the problem.

It is stated by many writers that as a rule the syphilis which precedes tabes has been benign, inasmuch as the secondary and tertiary symptoms have not been conspicuous. But exceptions to this rule are not very rare. It might be inquired whether an infection which is followed by metasyphilis should be considered benign, and whether the condition of the majority of those infected is not the same as that of those patients who develop tabes at a later period. This question could be best decided by the syphilologists. Fournier believed that tabes so often follows apparently benign cases of syphilis because the antisyphilitic treatment has been insufficient in such cases. This conclusion is manifestly open to criticism, and Fournier himself has admitted that those whose treatment has been "sufficient" according to his meaning of the word do acquire tabes. The further question how many of the infected subsequently develop tabes cannot yet be answered. Here, too, the syphilologists could be of assistance, but their conclusions should not be based on observation of hospital cases. It is certain that only a small part of the syphilitic become tabetic. Consequently there must be reasons for the selection—accessory conditions or causes. We may suppose that chance often plays a part, just as no especial explanation is demanded of the fact that one patient has, another has not an iritis or an orchitis syphilitica. In any case, very little is known as yet as to the accessory conditions.

Accessory Causes.

The most natural assumption is that the parts which functionate most actively are especially threatened. With regard to tabes the following point seems to me worthy of note. The functional activity of the nervous system, especially of its sensory portions, is in a certain sense proportional to the development of civilization. We may therefore suppose that the danger of tabes grows as civilization advances, thus explaining the statement made in many quarters that syphilis is frequent but tabes rare among certain uncivilized peoples. It is also remarkable that notwithstanding the frequency of hereditary syphilis tabes appears to be very rare in youth. Neuropathic condi-

tions may also be regarded as an accessory cause. The neuroses also increase with the progress of civilization and perhaps more rapidly than the latter.

From my own experience the importance of heredity in tabes seems to me to be very slight. I was probably the first to call attention to this point (1879). Among 61 tabetic cases I found only 1 case in which a member of the family had had tabes and only 6 in which other nervous affections (paralysis, epilepsy, and nervousness) had appeared in the family. At a later time Charcot and Strümpell expressed similar opinions. Erb found in 279 patients 77 neuropathic, and only twice an apparently direct hereditary transmission—that is, both father and son affected. Twice he found two syphilitic brothers with tabes. Charcot's pupils alone maintain that nervous heredity plays an important part. Ballet and Landouzy found in 101 cases no known cause 52 times, certain syphilis 14 times, probable syphilis 11 times, undoubted nervous heredity 17 times, probable heredity 7 times. The slight value of these statistics appears from the small number of cases of syphilis. Under the term, nervous heredity, are comprehended all possible nervous diseases of the family. But I will undertake to find among 100 patients of any kind 24 in whose family nervous diseases of some kind have occurred. It is possible, nevertheless, that the neurotic, if they become syphilitic, are in greater danger of developing tabes than others, their nervous system being, so to speak, a *locus minoris resistentiæ*. The fact that two or three cases of tabes or of general paralysis occur sometimes in the same family seems to point to this. But such theories are pure assumptions and it is certain that in the majority of cases of tabes heredity plays no part.

That the abuse of alcohol is to be regarded as an accessory cause of tabes I do not believe. It is but little considered in the statistics, for I find it mentioned in only about ten per cent. of the cases. It would probably be found that ten per cent. and more out of one hundred men of the well-to-do classes who are not tabetic have indulged in alcoholic beverages to excess. However justifiable the opposition to alcohol may be, it should not always be made a scapegoat.

That the misuse of tobacco has anything to do with the causation of tabes is a quite unproved and highly improbable assertion.

Exposure to cold is certainly not without importance. It was formerly regarded as the chief cause of tabes and there are still authors who so regard it. The patients themselves are apt to be much in favor of this etiology. Sometimes they feel pains in the legs which they and their friends regard as rheumatic. Hence they draw the conclusion: "Since I have rheumatic pains, I must have

taken cold." At other times the patients can really point to such exposure which has preceded the first appearance of the pains. In the latter cases it is natural to suppose that tabetic changes were already present, but that the exposure to cold was the exciting cause of the first pains, just as, when tabes already exists, attacks of pain are not infrequently produced by cold. A greater importance is to be ascribed to what may be called chronic chilling, living in damp dwellings, working in the water, bivouacking in the field, etc. It is conceivable that many a syphilitic would have escaped tabes if he had not been exposed repeatedly to cold. On the other hand, countless men endure such exposure without material injury, and in the greater number of tabetic patients there is no history whatever of it.

More difficult of credence is the idea that overexertion is of influence in the causation. Almost all men are exposed to fatigue, but tabetic patients are often less so exposed than many others. That overexertion may be prejudicial to one who is already ill is evident enough.

Much attention has been given to an examination of the question whether there is a traumatic tabes. Hitzig has undertaken the task of examining the cases reported under this title, and out of thirty-five cases has selected six which could to some degree withstand criticism. Even in these cases the proof that tabetic changes were not present before the traumatism is generally wanting. That an injury alone could produce tabes is so unreasonable that it is not necessary to discuss it seriously. At most it could only be supposed that the effect of the injury was to localize or make worse the original disease. It is not infrequently stated that the tabetic pains first appeared in an injured limb. Tabes is of course sometimes combined with traumatic hysteria.

It would be a waste of time to discuss all of the various things which have been brought forward as causative of tabes, such as sexual excesses, spermatorrhœa, puerperal processes, hemorrhages, acute diseases, emotional disturbances, etc. These are of course all injurious to and diminish the power of resistance of the body and so may favor the development of any disease, tabes among others, but they cannot be conceded to bear any direct relation to the tabetic process.

When it became evident that in the majority of cases tabes patients are syphilitic, and that tabes is only intelligible as the result of an intoxication, many thought that perhaps other poisons besides that of syphilis may occasionally be the cause of tabes. The observations of Tuczek, who found in ergotin-poisoning a chronic disease with some tabetic traits and with degeneration of the posterior columns, seemed especially to point towards this view, and the name

"ergotin-tabes" was employed. That the affection in ergotin-poisoning is neither clinically nor anatomically genuine tabes Tuczek himself has shown, and no one as yet has been able to find a poison which can cause that disease. It is highly improbable that such a poison exists, for in all pathology there is no example to show that two poisons can produce precisely the same disease, least of all a disease so *sui generis* as tabes.

PATHOLOGICAL ANATOMY.

The clinical manifestations of tabes are those of a slow death of nerve fibres and cells. It is quite certain that the first change is the death of "the nobler parts," the parenchyma, and that the implication of the interstitial tissue and of the blood-vessels is secondary. The explanation of the process probably is that, through the death of the parenchyma, the impulses to growth latent in the subordinate tissues are liberated, and that the products arising from the retrograde changes of the parenchyma act as irritants to cause inflammatory proliferation. The secondary changes reach a different degree of development in different cases, this depending presumably in great part upon the rapidity of the course of the disease, perhaps also upon individual peculiarities. The parts first affected in tabes (aside from certain localities in the brain) are the fibres which originate in the spinal ganglia and run their course through the posterior columns of the spinal cord, the fibres of the posterior roots. It appears that the first changes of these fibres do not always become visible at the same places, and that the degeneration is sometimes first found in the root zone of the posterior columns, sometimes in the posterior roots themselves. The cells to which the root fibres belong are those of the spinal ganglia. The latter present at first little evidence of disease; they have been found in some cases apparently normal, in others partly degenerated. The cell and its processes constitute a physiological unit, and this unit is either healthy or diseased. Whether the change manifests itself in the cell or in the processes, the presence of either form proves that the whole is diseased.

The visible changes, as is well known, propagate themselves in definite directions, *i.e.*, towards the ends of the processes. The degeneration of the processes of the posterior roots is followed by a similar degeneration of their continuations in the spinal cord. Hence the distribution of degenerated fibres in the cord indicates the course of the root processes. The changes in the posterior column in tabes are very similar to those produced by section of the posterior roots. The ascending root fibres lie first in the columns of Burdach, pass

inwards at a higher level and form the columns of Goll. Since, as a rule, the roots of the lumbar cord are first affected in tabes, there is found in the first stages of the disease a degeneration of the columns of Burdach in the lower part and of the columns of Goll in the upper part of the cord, and also, in the vicinity of the affected roots, a degeneration of the entering root fibres. As the disease progresses, cells of the spinal cord and their processes become affected and the degeneration attacks the gray posterior cornua and the columns of Clarke as well as the bundles of fibres which proceed from them. The appearance of a cross-section of the cord must accordingly vary with the height at which it is made and with the duration of the disease. A further cause of difference is that at the outset the same fibres of the posterior roots are not always affected and that the diseased roots are not attacked with equal severity. Clearly the anatomical picture will differ if the roots of the lumbar or, as happens in rare cases, of the cervical cord are alone affected from that seen if the dorsal and cervical portions are involved simultaneously with the lumbar portion. Yet the changes are always approximately symmetrical. In cases of tabes of long standing the whole posterior columns, except a small tract beside the posterior commissure, are degenerated. They appear to the naked eye diminished in size, in transverse sections sunken and gray. Hence the designation formerly occasionally used for tabes—gray degeneration of the posterior columns. The microscope shows that the little discs which represent the nerve fibres in cross-sections have disappeared and that the tissue consists of wavy fibres—the proliferating glia. Occasionally the pia spinalis is opaque and thickened over the posterior columns and sometimes a narrow band of degenerated tissue surrounds the cord—marginal degeneration. The vessels are often but little changed, sometimes their walls are thickened, as is seen also in descending degeneration due to cerebral lesions.

Morbid changes are found in tabes not only in the posterior roots and in the posterior half of the spinal cord, but also generally in the brain and in the peripheral nerves.

In the brain the cerebral nerves and their ganglia are especially affected. The olfactory nerve is rarely implicated, although Pierret found it quite atrophied in a case in which during life there had been first delusions of smell and later anosmia. The weakness of sight or the blindness of the tabetic is due to gray degeneration of the optic nerve. The layer of ganglion cells of the retina is supposed to correspond to the spinal ganglion. The nerve fibres of the optic nerve appear to be first changed, but sooner or later the cells of the retina are also affected. The process then advances centripetally, towards the

corpora quadrigemina. The paralyses of the eye muscles as a rule are due to degeneration of the nerve nuclei in the vicinity of the third ventricle or to degeneration of the nucleus of the abducens. In the nuclei the fine fibres between the cells have disappeared, the cells themselves are in part shrunken and pigmented, in part have also disappeared. At the same time with the implication of the nuclei there is found degeneration of the root fibres and of the peripheral nerves of the eye muscles. In a few cases only the latter or some branches of the latter have been found degenerated, the nuclei not being affected. In the trigeminus the Gasserian ganglion corresponds to the spinal ganglion. From it arises the ascending spinal root of the trigeminus. This is found atrophied, if trigeminal symptoms have existed during life, and wasting of the Gasserian ganglion has also been demonstrated. The other cerebral nerves are similarly affected, but with regard to them there are but few observations on record. In tabetic deafness the auditory nerve has been found atrophied. In paralyses of the larynx sometimes the nucleus of the vagus, sometimes only the peripheral nerve appears to be degenerated. In hemiatrophia linguæ degeneration of the nucleus of the hypoglossus of the same side has been found.

The lesion which is the cause of reflex iridoplegia is not yet known. It has been sought in the gray matter surrounding the third ventricle, and evidently it must lie in the neighborhood of the iris nucleus. But as it must be very minute it is not easy to find, and if extensive degeneration is found in the midbrain one does not know how to distinguish this nucleus.

Even when no pronounced general paralysis has existed, not infrequently slight changes of the cerebral cortex have been found which in appearance and locality corrrespond to those of this paralysis. They are probably to be so regarded, a slight mental impairment having existed without attracting attention.

Changes in the peripheral nerves appear to be present pretty regularly. The nerves of the skin are found to have degenerated, and in more advanced cases a part of those of the muscles as well. In diseased bones the bone nerves have been seen to be degenerated, and in the vicinity of the perforating ulcer of the foot and of similar trophic disturbances the nerve fibres are generally completely destroyed. As a rule, the number of the degenerated fibres diminishes as the nerve ascends. In the large nerves, only a relatively small number of degenerated fibres are usually to be found, and notwithstanding the degeneration of the muscle nerves the anterior roots are normal.

Taking all these facts into consideration we must say that there exists in tabes a widespread degeneration of the nervous system, but

that the parts which subserve sensation are most regularly and most seriously damaged. The centripetally directed process of the spinal-ganglion cell degenerates throughout its entire extent, the peripheral process, *i.e.*, the sensory nerve, especially in its most distal portions. It is evident that such a disease is correctly called a system disease, for systems, *i.e.*, nerve structures which together perform a distinct office, are particularly attacked by it. Among other things, the reflex iridoplegia is a proof that tabes is a system disease, for only a selective process could reach constantly with infallible certainty the minute spot in the brain which controls this singular symptom. It is surprising that in opposition to these clearly ascertained facts even modern authors have hit upon the strange idea of explaining tabes by mechanical conditions, of thinking that a meningitis could compress the posterior roots, and other similar absurdities.

If the question is asked, how far the individual symptoms of tabes can be referred to the degeneration of definite nerve fibres, only an unsatisfactory answer can be given. The fibres of the posterior roots propagate stimuli centripetally. Disease of these is the most important element in tabes. To this is due the fact that the most serious disturbances are those of sensibility and of reflex excitability. The pains, the paræsthesiæ, the anæsthesia, the abolition of the tendon and pupil reflexes, the relaxation of the muscles, the ataxia, the disturbances of the bladder, of the intestine, and of the sexual organs may all be referred without difficulty to disease of the centripetal fibres. In details, however, difficulties arise, since often we cannot explain the disease process by the anatomical findings nor is its anatomical location always evident. If we say that the pains are due to an irritation of the fibres of the posterior roots, the words do not help us to conceive how the irritation originates. Especially is the occurrence of the pains in paroxysms difficult to understand. As a matter of fact not only the attacks of pain and the crises of tabes in general are incomprehensible, but also epileptic convulsions and all similar "discharges." The use of the word "discharges" seems to suggest the idea that an explosive material collects in the diseased nervous tissue which when it is present in sufficient quantity explodes on any occasion.

The partial anæsthesia also presents difficulties to our understanding. Relying upon old experiments upon animals, the value of which is quite dubious, and upon the fact that in definite diseases of the gray matter of the spinal cord (especially syringomyelia and hæmatomyelia) partial anæsthesiæ occur, many are inclined to make the lesions of the posterior cornua responsible for the analgesia. Opposed to this is the fact that the analgesia often appears early, while the degenera-

tion of the gray matter is demonstrable only rather late in the disease; also the fact that analgesia occasionally occurs in neuritis likewise. If there were especial fibres for pain—which I do not believe—the analgesia could be easily explained.

Why in different cases the anæsthesia presents itself quite differently, not only in its location and extent but also in its form, why sometimes retardation of sensation, after-sensations, etc., can be demonstrated, sometimes not, we do not know. Nor do we know whether it makes any difference if degeneration is found in the fibres of the peripheral sensory nerve or only in the other process of the spinal-ganglion cell, the fibre of the posterior root, whether the implication of the ganglion cells in the degeneration is of importance clinically, whether the lesion of the sensory paths in the gray substance has a different effect from lesion of the peripheral nerves, etc.

It is much more easy to decide the question as to the height of the lesion, when certain symptoms are present. The answer is really given by normal anatomy, in so far as this can inform us through what roots the individual portions of the body are connected with the central organ. Yet the knowledge we at present possess is partly based upon the teachings of pathology. We know that the nerve fibres which come from the bladder enter the spinal cord through the posterior roots of the sacral cord, and that the sensory fibres from the legs belong partly to the sacral, partly to the lumbar cord. Thorburn and others have attempted to define the regions of the individual roots. The fibres the injury of which causes the disappearance of the patellar reflex enter the cord with the third or fourth lumbar nerve. Westphal has taken the pains to ascertain accurately the part of the spinal cord the degeneration of which causes the loss of the knee phenomenon. In the trunk and arms our knowledge as to the root regions is more accurate than in the legs. It is unnecessary to enter into this subject in greater detail here. In the older text-books statements are found to the effect that the ataxia is connected with disease of the funiculus cuneatus and the vesical disturbances with disease of the columns of Goll. These statements our present knowledge shows to be without foundation.

Symptoms.

However numerous the symptoms of tabes may be, and however varying may appear the picture of the disease, there are yet a few essential symptoms which are always present, upon which the diagnosis depends, and which by themselves may constitute tabes. These essential symptoms are also generally the first to appear, and it is they which characterize tabes incipiens. They are reflex iridoplegia,

lancinating pains, disappearance of the knee phenomenon, vesical disturbances, and certain disturbances of sensibility.

Reflex Iridoplegia.—This symptom was first pointed out by Argyll Robertson and is therefore often called by his name. It consists in this, that the pupil does not contract when light strikes the eye, although it contracts rapidly and strongly upon convergence. It is therefore to be carefully distinguished from paralysis of the iris. In the former only the reflex contractility is absent, the motility remaining perfect; in the latter motility in general is lost. To demonstrate reflex iridoplegia, therefore, it is never sufficient to prove that the reaction to light is wanting—a good reaction on convergence must be shown to exist. Paralysis of the sphincter pupillæ is observed in various diseases, reflex iridoplegia is found only in tabes and general paralysis. I say "only," but strictly speaking that is incorrect, for in very rare cases of focal lesions of the brain, especially of the region of the corpora quadrigemina, reflex iridoplegia has been observed. But these cases are so rare, and there is so little reason to fear that they will be confounded with tabes, that in practice the above statement is correct. Some authors state that they have found reflex iridoplegia occasionally in alcoholic subjects and in other patients. It is, however, obvious that if one alcoholic subject out of one hundred has reflex iridoplegia, this is much more probably a symptom of tabes than of alcoholism. For we know that reflex iridoplegia may precede the other signs by years, and with the prevalence of tabes it is to be supposed that among one hundred drunkards one or more will be found with incipient tabes. Since reflex iridoplegia is found in tabes only, since in the majority of cases it is the first sign of tabes, and since it is objective and easily demonstrated, it is the most important symptom of tabes.

Reflex iridoplegia is found in three-fourths of the cases of tabes. In 1880 Erb examined 84 tabetic patients. Of these 59 had reflex iridoplegia, 12 very sluggish, weak, and imperfect reaction to light, and only 13 had a normal light reaction. Similar figures have been obtained in subsequent investigations. In some cases of tabes reflex iridoplegia is absent although the disease may have a duration of years. If it appears at all, it is generally present from the first. As has already been said, it may exist for years as the only symptom. If it alone is found, the diagnosis of tabes (or of general paralysis) may in my opinion be made; but it should be added that the time at which other symptoms are to be expected cannot be foretold. These are, however, exceptional cases. As a rule, the patient comes to the physician complaining of some of the symptoms of tabes. The first thing that the latter should do is to examine the pupils. If he finds

reflex iridoplegia the diagnosis is made; if he does not find it, further investigation is necessary.

The best way of testing for reflex iridoplegia is to place the patient near a window so that he can look out of doors. The physician, standing before him, covers the patient's eyes (which remain open) with his hands, waits ten, twenty, thirty seconds and taking away his hands observes one of the patient's eyes. Care must be taken lest the light reflected from the cornea interfere with the view of the eye. The examination is easier when the iris is light-colored and the pupil is wide. If positive results are not obtained by this method the examination may be repeated in a dark room, and the light of a lamp or light from a chink in the shutters may be allowed to fall obliquely upon the eye. If necessary a lens may be used. Of course in every method of examination care must be taken that the patient looks into the distance and does not alter his accommodation. If the pupil remains immovable under illumination, *i.e.*, if it is paralyzed for light, the test for contraction on convergence should follow. The most simple way of doing this is to have the patient look at his own nose. Or he can be requested to look first out of the window and then into the face of the examiner or at his finger. If the patient cannot converge his eyes (in paralysis of the ocular muscles) the convergence reaction is apparently absent. If the latter is absent in spite of convergence the iris is quite paralyzed and then in the majority of cases some form of ophthalmoplegia will be found to be present. In the later stages of tabes entirely paralyzed pupils may also be found without ophthalmoplegia, but not very often. In any case the complete immovability of the pupil is a less characteristic sign of tabes than reflex iridoplegia.

Reflex iridoplegia is generally bilateral, in rare cases unilateral. In the latter, the light reaction is wanting not only under direct illumination but also under illumination of the other eye; while the pupil of the other eye reacts both under direct illumination and under illumination of the eye with paralyzed iris, *i.e.*, the latter has lost not only the direct but also the "consensual" reaction. The "paradoxical" pupillary reaction—that is, the apparent dilatation of the pupil when light falls upon it—has been described as an especial form. The condition in these extremely rare cases is really reflex iridoplegia. When the covering hand is removed the pupil at first is unchanged, but after a few seconds it dilates somewhat. The explanation of this is that the patient exerted his accommodation while his eyes were covered; when the accommodation relaxes again, the pupil dilates. As has been said, the paradoxical reaction is very rare.

Slow and imperfect light reaction is to be distinguished from reflex iridoplegia. Careful observation is necessary here, for not every weak reaction is abnormal. To constitute abnormality there must be a distinct difference between the light reaction and the convergence reaction. The case is clear when the sluggish reaction is unilateral. To properly discriminate the shades of difference in the pupillary reaction practice is necessary.

Associated with reflex iridoplegia is generally a loss of pupillary dilatation after painful stimulation. When the skin is pinched or a sudden noise is heard the pupils of the healthy subject dilate, those of the tabetic remain unchanged. Even during the violent, lancinating pains of tabes the pupils generally remain contracted. Not only the reflex contraction but also the reflex dilatation is consequently absent in tabes. We may say that the pupillary reflexes are abolished. However, the examination for reflex dilatation has hardly any practical value. It is also hardly ever necessary to test the pupils of the tabetic by means of drugs. Atropine dilates the pupils in tabes, but more slowly and feebly than in health.

Very often, besides defective reaction, there is also present an abnormality in the size of the pupils. Most frequently there is contraction of the pupils in tabes—myosis, which, as a symptom of tabes, has also been called spinal myosis. Of forty-seven tabes patients of Erb's with reflex iridoplegia thirty-seven had pronounced myosis. But reflex iridoplegia and myosis are two independent symptoms; they may occur together or separately. The rule is that in time myosis is superadded to the reflex iridoplegia. Myosis without reflex iridoplegia is also met with in tabes, but not often. The degree of the myosis varies of course. The pupils are often very much contracted, so much so that their appearance attracts attention at the first glance at the patient. Much more rare is mydriasis, but it does occur, especially at the beginning of the disease. It may be unilateral or bilateral and occur with or without reflex iridoplegia.

In moderate degrees of unilateral myosis or mydriasis one may be in doubt whether the dilated or the contracted pupil is the abnormal one, and sometimes this cannot be determined, but it is not a matter of importance. Difference in size of the pupils formerly played a more important part than at present in the diagnosis. Yet such difference is of great value as a danger signal, so to speak, which immediately attracts attention and demands further investigation. Small differences are of importance only if they arise during the examination; larger differences always constitute an important sign, but the reaction is here still decisive.

Occasionally the tabetic pupil is not circular but elliptical or ir-

regular in shape. This deformity is, of course, to be distinguished from that due to synechiæ.

The Lancinating Pains.—Tabes almost always first makes itself known to the patient by pains. These are sometimes slight, sometimes severe, sometimes frequent and sometimes infrequent, but are probably never entirely absent. They are described by most patients as resembling the pain caused by a stab or an electrical shock; they begin suddenly and last a short time, but return after an interval. As in trigeminal neuralgia, the attacks of pain are composed of groups of single pains and last a day, a night, or some days and nights. Between the groups there may be minutes or hours of freedom from pain. In an attack the location of the pain either remains the same or the pain appears in different places. The former seems to me to be the more frequent. The pain rarely corresponds to the course of a nerve; it is generally described as being in the flesh, or in a part of the foot, as in the little toe, sometimes it is assigned to a joint or to the skin. With deep as with superficially situated pains the skin over the painful spot may be hypersensitive so that the patients avoid touching it. This sensitiveness often lasts some time longer than the attack of pain. Many patients also state that the pain bores through their limbs longitudinally. If the pain appears in the trunk it generally presents itself as unilateral, more rarely bilateral girdle pain; occasionally only certain points in the back or in the chest are painful. In the head the pain may be very similar to the common trigeminal neuralgia. Besides this most frequent form, in describing which the expression "lancinating" or "fulgurating" is in place, other kinds of pains are more rarely observed, which are described as constricting, boring, or burning, as if the foot lay in an iron vise or as if a sharp iron or a glowing coal were thrust into the flesh or into the bones.

As has been remarked, the severity of the pains varies greatly. Some patients state that they have had no pain, and only when closely questioned do they admit having had slight pains. The majority have an attack of pain every few weeks or months. Unfortunately there are not a few who are tortured almost incessantly with pains, or at all events have more days with than without pain, so that the designation "tabes dolorosa" may be used. The pains may be so severe that even strong men writhe and cry out under them.

Exposure to cold and "changes in the weather" are often regarded as exciting causes. Sometimes digestive disturbances seem to play a part in the causation. In rare cases suggillations, which run the course of an ordinary bruise, or groups of herpetic vesicles appear at the location of the pains.

The Absence of the Knee Phenomenon.—This sign has this in common with reflex iridoplegia, that it is an easily demonstrable objective symptom, but it is by far not so characteristic, since any neuritis may cause the disappearance of the patellar reflex. It disappears in the great majority of cases of tabes and at a very early period, but its absence must not be regarded as essential for the diagnosis, since cases of tabes with preservation of the knee-jerk are not so very rare, or at all events characteristic symptoms may be present before the patellar reflex disappears. It is best to test for the knee phenomenon by having the patient seated with the foot resting by the entire sole upon the floor, in such a position that the leg forms an angle of about 60° with the thigh. The examiner places his left hand upon the patient's quadriceps and with the percussion hammer held in his right hand strikes a quick hard blow upon the middle of the ligamentum patellæ. One leg may also be crossed over the other, or the patient may sit upon a table and let his legs hang free. If the patient is in bed the examiner's left hand may be placed under the knee, raising it until the leg and thigh form an angle of 60° to 70°. If the contraction of the quadriceps is distinct, we may content ourselves with the examination of the limb through the clothing. If, however, the knee phenomenon appears to be absent, it is well to make certain of the fact by repeating the examination with the limb unclothed. It is always necessary for the patient to relax his leg muscles. This is facilitated by the so-called Jendrassik's method, which consists in the patient's interlocking the finger of his hands and then pulling strongly as if he would tear his hands apart. The fists may also be clenched or the palms of the hands pressed against each other, etc. With practice it is easy to tell if the knee phenomenon is absent in tabetic patients; the percussion of the ligamentum patellæ produces a peculiar "dead" sensation, for the elasticity of the ligament has disappeared.

The patellar reflex does not usually disappear suddenly in tabes, it first becomes weak. If an examination is made at that time a barely perceptible contraction of the quadriceps is detected, or on repeated examinations the knee phenomenon is demonstrable at one time but not at another. It also occasionally first disappears or becomes weak in one leg only. Such differences are always suspicious and demand further investigation. Transitory increase of the knee-jerk may occur at the beginning of tabes, but this is rare. If the tendon reflexes are increased, the question always arises whether the case is not one of general paralysis with tabetic symptoms.

If the knee phenomenon is absent in tabes the other tendon reflexes of the legs are also abolished. The tendon reflexes of the arms may

also be absent at an early, but more frequently at a late stage of the disease. Occasionally in the so-called tabes descendens the disappearance of the knee phenomenon may follow that of the triceps reflex.

Vesical Disturbances.—These are very important diagnostically, since in tabes they are the rule, in neuritis the rare exception. But they must be inquired for, since they are often slight, in which case the patients are not apt to mention them. Sometimes the patient is obliged to wait longer than formerly before the urination commences, the stream becomes feeble and straining is necessary to evacuate the bladder; sometimes, on the other hand, the evacuation takes place too easily, so that the patient is obliged to hurry when the call to urinate occurs, and occasionally a little urine flows away during coughing, laughing, on sudden changes of position, and in sleep. More serious disturbances in the first stages of tabes are rare. Retention and true incontinence of urine occur only exceptionally; in the later stages they are frequent. Sometimes the patients regard the bladder trouble as the chief disease and on that account go to a surgeon, who if he recognizes their true nature sends the patients to the neurologist.

Leimbach has recently published interesting statistics as to the frequency of the signs of tabes discussed above. They are derived from Erb's material. Of 400 tabes patients, in 92 per cent. the knee phenomenon was absent; in 4.25 per cent. it was present, but abnormal (together, 96.25 per cent.); 88.25 per cent. had lancinating pains; 80.50 per cent. had vesical disturbances; 70.25 per cent. had changes in the pupillary reaction, and 48.25 per cent. had changes in the size of the pupils. The tabes had begun with lancinating pains in 67 per cent. and with vesical disturbances in 22.5 per cent. Of 100 tabes patients whose disease had had a duration of from one to two years there were changes in the tendon reflexes of the legs in 100 per cent., and in the pupillary reaction in 63 per cent.

Disturbances of Sensibility.—Under this head I include paræsthesiæ and anæsthesia, as well as the disturbances of motility dependent upon them.

Paræsthesiæ are often among the earliest signs. The patients complain especially of paræsthesiæ in the legs and in the region supplied by the ulnar nerve, and of the so-called "girdle feeling." They say that they have the sensation as if the soles of their feet were "asleep," or as if they walked upon cotton or fine sand. Ants appear to run over the skin of the legs (formication); there is a peculiar sensation of numbness. A burning sensation is rare. Very often it is said that the feet or the feet and legs are unendurably cold. Sometimes the paræsthesiæ first manifest themselves in the region of the buttocks or in the sexual organs.

The girdle feeling is generally first felt below the navel. In the course of the disease it may advance upwards, or it may from the first feel like a band about the chest. It does not always completely encircle the trunk; it may be unilateral, or the patients may describe the sensation as the pressure of a flat surface upon the chest or in the pit of the stomach; sometimes it appears to them as if a constricting girdle were really placed about them, sometimes there are simply undefined painful sensations.

The ulnar paræsthesiæ manifest themselves mostly as formication or numbness of the fourth and fifth fingers. Many patients have such sensations on the ulnar surface of the forearm as far as to the elbow.

Much rarer than the forms already described are paræsthesiæ of the face. The feeling may be as if the face were covered with cobwebs or as if a mask were stretching and compressing the skin of the face. Charcot has called this singular symptom "Hutchinson's mask." I have seen it once as the first symptom of tabes. Other trigeminal symptoms are often present with it.

In testing the sensibility, in spite of the paræsthesiæ, no so-called objective disturbances of the sensibility may be detected. As a rule, however, they are to be found. For a clinical examination of the skin it is amply sufficient to test the sense of touch by light contact with the finger or a small brush, to test the sense of pain with a needle, and the temperature sense by means of cold and warm objects.

In the legs of the tabetic there are generally found at the outset lessened sensitiveness to pain and either slight or no diminution in the sense of touch. The latter is first found diminished on the soles of the feet. Elsewhere hypalgesia decidedly predominates. Only moderately severe pricks with a needle are felt to be painful; at a later period pricking is not felt as pain at all, only as a sensation of pressure; a fold of the skin can be pierced without the knowledge of the patient. As a rule, as time goes on the disease progresses, the region of hypalgesia advances upwards from the feet, but a certain degree of anæsthesia is often found at an early period in the buttocks and their vicinity. Sometimes the hypalgesia appears in irregularly bounded and irregularly distributed patches. On the borders of the hypalgesia or between the hypalgic zones, it is said that hyperalgesia is often found. Sooner or later, in addition to the hypalgesia a diminution of the sense of touch is also manifested. The temperature sense may be apparently normal for a long time, but supersensitiveness to cold is common. In cases of long standing there may be complete anæsthesia of the legs, or even of the greater part of the body, but in very many cases the anæsthesia remains incomplete to the end. M. Lähr has demonstrated a diminution of sensibility for gentle contact

in the trunk of tabes patients at an early stage of the disease. It generally first shows itself beneath the breast, then near the scapula, and finally it forms a zone which completely surrounds the body, and the symmetry of which is more pronounced along its upper than along its lower border. Sensibility for pain appears at first not to be affected; at a later time it diminishes. The temperature sense is also long preserved. If the anæsthesia involves the arm, it first shows itself in the axilla, then appears on the ulnar side of the arm, and finally on the radial side. The anæsthesia appears in the fingers with a certain independence. It sometimes involves all the fingers, sometimes only the ulnar, sometimes the median, or with apparent capriciousness it may attack only a certain number of fingers. Generally there is diminution of the sense of touch with hypalgesia. On the head, parts or the whole of the region of the trigeminus, including the mucous membranes, may be hypalgic, hyperæsthetic, or rarely quite insensitive. Marked hyperæsthesia is everywhere rare, aside from that which accompanies the lancinating pains, but slight hyperæsthesia or hyperalgesia may occur in limited areas.

The so-called *retardation of the conduction of pain* is very frequently combined with the analgesia. The patient does not feel the prick at once but only after one-half, one, two, or more seconds. Leimbach found this retardation of the sense of pain in 36.5 per cent. of tabes patients. Sometimes there is a so-called double sensation, *i.e.*, the patient feels the prick as a touch and only afterwards as pain. Occasionally a retardation of the sense of touch also is found, contact being at first not at all perceived, but subsequently correctly located. A painful after-sensation also occurs, *i.e.*, the pain of the prick lasts much longer than would be expected, or the prick may not be felt at all at first, but after a time the patients complain of a violent and continued pain. Polyæsthesia and allocheiria are rare phenomena which have been described. In the former instead of one touch two or three are felt; in the latter a touch upon the left limb is felt upon the right, or *vice versa*. In case of both of these symptoms there is probably a psychical perturbation—incorrect appreciation of indistinct perceptions.

Insensitiveness of the deeper parts appears to be both earlier and more frequent than that of the skin, but is more difficult to demonstrate. Diminution of the muscular sense is often spoken of, but in reality the condition appears to be a diminution, in the first place, of the joint sensations and then diminution not only of those of the muscles but also of those of the tendons. In testing for these we see if the slightest movements of the joint, which the healthy subject perceives, are still felt. While the patient's eyes are closed, we place

his limbs in certain positions and have him describe them or imitate them with the other limb. Or we have him execute prescribed movements, or have him estimate weights by movements, etc. The simplest and most trustworthy test for disturbances of sensibility in the legs, which are especially important in tabes, consists in requiring the patient to stand still with closed eyes. If then his joints and other tissues are not normally sensitive, he cannot completely maintain his equilibrium, he totters. Unsteadiness when the eyes are closed, or Romberg's symptom, is an important and early symptom of tabes. Leimbach found it in 88.75 per cent. of tabes patients and in eighty per cent. of the patients in the first two years. If it is very marked, the patient cannot keep himself upright when he closes his eyes, but falls down. But in the beginning of the disease Romberg's symptom is detected only when the patient stands with his feet close together. We may also see if the patient can stand upon one foot with closed eyes, but it must not be forgotten that this is not possible for many healthy individuals. Romberg's symptom is generally noticed during the examination of the pupils. The patients have often perceived it themselves. They state that they are unsteady in the dark or that they totter if they dip their face in the basin when washing themselves.

If the insensitiveness of the deeper parts increases, movements in general become uncertain. This is called the *ataxia* of tabes. There has been much dispute as to whether ataxia depends upon anæsthesia or not. As far as tabes is concerned, it seems to me certain that the ataxia is simply an effect of the anæsthesia, especially of the defective joint sensibility, with perhaps the aid of paræsthesiæ in the deep tissues. The process is essentially as follows: The patient having lost the necessary control over his movements supplied by the sensibility of the joints and other tissues, seeks to supply the lack by the aid of sight, and therefore when he walks follows the movements of his legs with his eyes; on the other hand, he seeks involuntarily to compensate for the disturbances of innervation by an increased expenditure of force, so that he kicks and flings his legs about.

To demonstrate slight ataxia of the legs in tabes the patient is required to execute a variety of movements, while standing up and while lying down, and first with open, then with closed eyes. He is told to march forward, to "about face," to walk a line, at the command to sit down and stand up again quickly, to descend steps, to bend his knees. Marked ataxia betrays itself, when the patient is recumbent, even by the simple raising of a leg—a movement which is not performed steadily but by jerks and with lateral deviations.

More delicate is the knee-heel test, *i.e.*, touching one knee with the

heel of the other foot, both heels being employed in this way successively. The ataxic does not succeed in touching his knee in this manner, or only does so after several unsuccessful attempts. It is also customary to direct the patient to describe a circle with his foot. Sometimes slight ataxia is more readily detected by the last-named series of exercises, sometimes by those performed during standing or walking. If the ataxia is pronounced, the familiar ataxic gait is produced. The patient walks with his body bent forward, in order to keep his eyes directed upon his feet. He flings forward his foot and lets it fall heavily upon the ground, striking first by the heel, and overextends his knees. If the ataxia increase, the flinging and stamping gait is disturbed by lateral movements, the steps become unequal, and finally the patient can no longer walk at all, because his legs fly about in the air and the unpurposed movements are in preponderancy. In this severe form of ataxia a marked anæsthesia is generally also present. The patient has no idea as to the position of his legs, if he cannot see them; he "loses them in bed." The fact is well known, that in baths which are much frequented by the tabetic, if several patients are bathing in the same tank, occasionally a leg is protruded from the water and no one knows whether it belongs to him or to his neighbor. No fixed relation, however, between the ataxia and the anæsthesia can be demonstrated, because the former is the individual reaction of the latter. The ataxia presents itself in forms varying with the idiosyncrasy of the patient. In women, for example, the stiff, flinging movements are almost never seen. Their movements are rather uncertain and awkward. The ataxia appears, as a rule, at a late stage in the arms, but in some cases the arms become ataxic before the legs. At first the more delicate movements become difficult —writing, sewing, etc. The patients feel themselves impeded in dressing especially, because their hands are incapable of performing the movements of buttoning and tying, if the eyes cannot follow them. Violent movements may also intrude themselves here, which interfere with the performance of every act. Ataxic movements of the muscles of the face, jaw, and tongue are very rare. In contrast with the ataxia on motion, the locomotor ataxia, a static ataxia has also been described. By this is meant that movements occur even when the patient desires to keep quiet, that when he sits the trunk sways, the extended foot or hand moves here and there. These movements pass into the spontaneous movements to be described further on, the athetosis of the anæsthetic subject. Static ataxia is not an independent symptom but merely the expression of a high degree of ataxia.

Just when ataxia will make its appearance in tabes cannot be predicted; as a rule years pass; in some cases it develops in the first

years of the disease, indeed exceptionally it may be among the very first symptoms. Formerly it was given undue prominence as if it were the chief symptom of tabes. The name "locomotor ataxia" is an expression of this opinion. It is true that ataxia is the most striking symptom; by means of it tabes patients may be recognized as such on the street, but many patients die without having become ataxic, and in my consulting practice the tabetic patients without ataxia decidedly outnumber the ataxic. Leimbach found 74.75 per cent. ataxic. In seventeen per cent. of the cases the tabes had begun with ataxia of the legs. But this statement is manifestly based on the history as given by the patient, and this of course is not always correct. It is to be especially noted that while generally the ataxia begins slowly and insidiously, sometimes it develops as an acute affection so that in a few months or even a few weeks the patient who was previously not ataxic becomes unable to walk. After such a violent evolution there almost always follows a retrocession in the symptoms so that gradually some of the disturbances disappear.

Some of the other phenomena of tabes are manifestly dependent upon loss of sensibility. With the changes in the sensibility of the skin the skin reflexes also change. In contrast with the tendon reflexes, these are generally well preserved at the outset. They disappear only when the anæsthesia is marked. The state of the sole reflex in hyperæsthesia of the sole varies; generally it is simply diminished, but sometimes it is absent. If there is retardation of sensation of pain, a contraction of the foot sometimes first occurs simultaneously with the exclamation of the patient, which is manifestly an involuntary movement, but of cerebral origin, not a spinal reflex. Occasionally, however, a contraction follows a puncture immediately, and when the retarded sensation from the puncture is perceived the foot either remains motionless or twitches again.

More important than the results from the cutaneous anæsthesia are those arising from the loss of sensibility of the deeper parts. The involuntary or reflex muscular tension, generally known as "tonus," which is constantly present in the healthy subject, probably depends upon the fact that the central organ is properly served by the sensations, especially the joint sensibility. If the centripetal stimuli are deficient, the muscular tension, the tonus, relaxes. The extinction of the tendon reflexes is perhaps to be regarded as a manifestation of this phenomenon. The relaxation of the muscles of the tabetic often attracts attention through both the sense of sight and that of touch. The fact that in some tabes patients passive movements of great extent may be performed may depend in part upon the relaxation of the muscles and of the tendons, and in part directly upon the loss of sen-

sibility. If a healthy man lies upon his back, his knee being extended, flexion of the thigh cannot be carried far, for the subject soon feels pain and the flexors of the leg become tensely contracted; but in some tabes patients the leg can not only be brought to the vertical but can even be approximated to the trunk without material resistance and without causing pain.

Sometimes involuntary movements of the insensitive limbs occur, which must be distinguished from ataxia. Occasionally, for example, the toes of the recumbent patient are seen to execute slow movements of flexion and extension, which the patient cannot control or of which he is unconscious; or some fingers of the outstretched hand are flexed and extended or abducted and adducted, or the whole hand is raised and lowered. These and other similar involuntary movements are called spontaneous movements or, according to their character, athetosis or choreic movements.

Biernacki has seen many tabes patients in whom strong pressure upon the ulnar nerve at the elbow caused no pain, although healthy persons always feel pain from it. This observation has been confirmed several times. In France attention has been called to the insensitiveness of the testicle to pressure. The severe pain always caused in the healthy man by this procedure is often entirely absent in tabes patients, even strong pressure producing no painful sensation.

It is difficult to say how far the weakness of the legs of which the tabetic often complains depends upon the loss of sensibility. Certainly the legs, the muscles of which are relaxed and the movements of which are no longer regulated by normal sensibility, become quickly weary, and there is no doubt that a great part of the muscular weakness, which can frequently be objectively demonstrated, is thus to be explained. It is generally stated in the text-books that the strength of the patient is unimpaired, the incoördination only simulating muscular weakness. This is certainly not always true. In very many tabetic patients the movements are remarkably feeble. The fact exists, however it may be explained. After a time a certain degree of diffuse muscular atrophy is usually developed. Leimbach found that there was a feeling of weakness and that the legs easily became tired in 62.25 per cent. of tabes patients. In 19.5 per cent. weakness of the legs was the first sign of the disease.—

The Cranial Nerves.—The numerous other symptoms of tabes are difficult to classify. For the sake of simplicity we will begin with the affections of the cerebral nerves.

Symptoms on the part of the olfactory nerve are very rare. Occasionally disturbances of the sense of smell, such as loss or illusions

of it, have been described, but as yet it is not possible to make any trustworthy statements with regard to it.

On the other hand, one of the most important symptoms of tabes, not on account of its frequency but because of its significance for the patient, is atrophy of the optic nerve. Statements differ as to the frequency of its occurrence. Leimbach found it in only 6.75 per cent. of the patients. Gowers, on the other hand, met with it in fifteen per cent., while other statistics give about ten per cent. All are agreed that it develops almost always in the first stage of the disease, although exceptions of course occur. It seems that it may even be the first sign. In one case I saw it present when the pupillary reaction and the knee phenomenon were normal, disturbances of sensibility only developing a year later.

According to Leimbach it is the first sign in 1.5 per cent. of the cases. By the ophthalmoscope at first a paleness of the papilla, especially on the temporal side, is recognized, but the borders of the papillæ are sharply defined and the blood-vessels are unchanged. At a later period the papilla has a white color which may have a grayish, bluish, or greenish tinge. The veins then appear dilated; the arteries are, if anything, contracted. Notwithstanding the clinical differences, the ophthalmoscopic changes appear to be about equally diffused. The picture is often nearly the same in both eyes, although their vision may be quite different. In rare cases in spite of the amblyopia there is nothing abnormal to be seen in the fundus at first, visible changes in it only developing subsequently. More often the oculist finds the latter already present, before the patient has noticed a diminution in his power of sight.

Atrophy of the optic nerve manifests itself by amblyopia, color blindness, and contraction of the field of vision. Some patients complain of a mist which interferes with their sight; others simply notice that their eyes are failing. Generally the condition develops first in one eye, or at least is noticed in but one eye. But sooner or later the other eye is always attacked also. In fact it is very remarkable that a true unilateral atrophy of the optic nerve never occurs, although the other cerebral nerves may be affected on one side only. The difference between the eyes often persists for years; one eye may even be quite blind, while the other sees tolerably well. The final outcome, however, is blindness of both eyes. How much time will elapse before this occurs cannot be determined at the outset. Sometimes the amblyopia progresses rapidly at first and then remains stationary for a considerable period. It is well not to express a too unfavorable opinion, for many patients pass through some years with tolerable comfort. But there are malignant cases also, in which blindness results quickly.

Color blindness and contraction of the field of vision do not always keep pace with the amblyopia. Color blindness especially may appear first and in a certain sense independently. Green and red usually disappear first, then yellow and blue. Some patients see every thing gray in a gray field.

The field of vision is generally found concentrically contracted in amblyopia, but may also have jagged and irregular borders. Now and then the field of vision may remain approximately normal although the acuity of vision is much reduced. In a few cases hemianopsia has been found or defects corresponding to about a quadrant have been seen. Gowers thinks that since the ophthalmoscope reveals no corresponding lesions in the retina, a lesion of the chiasm is to be assumed as the cause. But there may be a complication present, an occipital focal lesion. Central scotoma is of very rare occurrence in tabes.

It is a singular fact that atrophy of the optic nerve appearing early in the disease seems to have an inhibitory effect upon the development of tabes. This was first shown by Benedict. Gowers, Martin, and others made the same observation later. It is quite certain that patients with atrophy of the optic nerve often have remarkably few symptoms, little pain and little anæsthesia, and hence no ataxia. The latter appears finally, but fifteen, even twenty years may elapse before its appearance. If, on the other hand, this atrophy occurs later, when ataxia is already present, there is no evidence of "inhibition."

The tabetic paralyses of the eye muscles constitute an interesting chapter. In the first place it is to be remarked that the majority of all paralyses of the eye muscles in adults are of a tabetic nature. This is not sufficiently recognized, first because these paralyses may be among the first symptoms, and secondly because they are very often transient. Almost every paralysis of the eye muscles brings the patient to consult the physician. If no other striking symptoms are present, a rheumatic or syphilitic paralysis is diagnosticated, and if the affection disappears in a few weeks, the physician regards the diagnosis as confirmed by the result of the treatment. It may be said that every paralysis of the eye muscles which develops without pain in a hitherto healthy person of middle age makes tabes very probable, for the paralyses of tertiary syphilis are almost always accompanied with pain, as are also the very rare rheumatic paralyses, *i.e.*, those dependent upon a neuritis of unknown origin, and recurrent paralysis of the oculomotorius nerve. Multiple sclerosis may cause paralysis similar to that of tabes, but is a much rarer disease. The sooner the tabetic paralyses of the eye muscles develop the

more transient are they. They last one or two weeks or one or two months. But if they have once appeared, their return is not improbable, and not infrequently these transitory forms pass finally into permanent paralysis. The initial paralyses often attack but one muscle, most frequently the externus, sometimes the internus or the levator palpebræ or the obliquus superior. The more extensive the paralysis the more probable is it that it will not entirely disappear. However, an almost complete ophthalmoplegia may disappear entirely. It is rather characteristic that even when the paralysis is limited, the other eye muscles often show signs of weakness. If, for example, an externus is paralyzed, it is often noticed that the other externus becomes fatigued when acting strongly, this being shown by slight nystagmus-like twitchings. Occasionally such twitchings are seen in all the extreme positions of the eye—a prodrome of ophthalmoplegia. Besides the double paralysis of the externi, double ptosis, a light ptosis in which only the upper part of the pupil is covered, is occasionally seen, and all forms of paralysis of the eye muscles may occur, such as paralysis of the motor oculi, unilateral or bilateral, and complete or incomplete ophthalmoplegia externa. Progressive ophthalmoplegia (or ophthalmoplegia externa) is the most important form of the permanent paralyses of the eye muscles in tabes. Hutchinson first described it. In it all the external eye muscles become gradually paralyzed, but the internal muscles are spared. The eyes are attacked simultaneously or in succession. The course of the asymmetrical affection is irregular and even at the last all of the muscles are usually not implicated to the same degree. The pupils are also usually paralyzed reflexly and are often also contracted.

Much rarer than paralysis of the external is paralysis of the internal muscles of the eye. This manifests itself either as ophthalmoplegia interna or as a paralysis confined to the ciliary muscle. In the former we find loss of accommodation and paralysis of the dilated pupil. Ophthalmoplegia interna occurs unmixed with other paralyses at the beginning of tabes and is then generally unilateral. If the history of the individual does not exclude the possibility of tabes, the presence of this paralysis renders it highly justifiable to suspect incipient tabes. The suddenly developed and transient unilateral loss of accommodation, first described by Fr. Müller, is also worthy of mention. In the later stages of tabes the paralysis of the internal muscles may supervene upon paralysis of other ocular muscles. The reflexly paralyzed pupil then becomes completely paralyzed, but generally does not dilate.

The pains, the paræsthesiæ, and the anæsthesia in the region of

the trigeminus have already been mentioned. So-called trophic disturbances may occur with the disturbances of sensibility, and also paralysis or atrophy of the muscles of mastication. Of the trophic disturbances especially to be noted is the tabetic shedding of the teeth. The teeth of the patient become loose and fall out without suppuration, generally even without pain. Often the patients pull out the loosened teeth with their fingers. One of my patients had thus cleared the entire upper jaw and carried the apparently normal teeth about in her pocket. Sometimes portions of the alveolar margins also become loose. The alveolar border always atrophies after the teeth fall out, and the lower jaw may become exceedingly narrow. The sensory trigeminus symptoms in cases in which the teeth are shed are sometimes marked, sometimes slight. In any case there is no direct relation between the disease of the jaw and the anæsthesia. In one patient of Vallin's there were only a tickling in the pharynx and the sensation of a lead cap upon the head; in another, only the left half of the face was anæsthetic and the muscles of mastication were atrophied only on the left side, although the teeth had fallen out on both sides. In a patient of Dolbeau's who had had such strong teeth that "he could crack one hundred and fifty apricot kernels in one day," all the teeth and a number of pieces of bone had fallen out without pain. In this case the atrophied jaws were covered with healthy mucous membrane and the cavities of the maxillæ were open so that the finger could be inserted into them; there was also a severe keratitis. Necrosis of the cornea, the so-called ophthalmia neuroparalytica, is very rare in tabes patients, but it does sometimes occur as this case shows.

Recently, tabetic ulcers of the mouth have been described and have been regarded as mal perforant of the mouth. As far as I can see, in all cases they have been ulcers which were connected with the loss of the teeth, and it may be surmised that their cause was the formation of sequestra. In L. Wickham's patient, for example, the teeth had previously fallen out, the alveolar process was in part destroyed, and the diseased tissues were insensitive; the sound encountered carious bone at the bottom of the ulcer, which was surrounded by an elevated margin and was covered with thin and ill-smelling pus. In other cases the maxillary cavities were open. In a case of Letulle's the mal perforant had destroyed the right half of the velum of the palate and a portion of the upper jaw; all the teeth of the upper jaw had been lost; the course was painless throughout. Tabetic salivation may also perhaps be regarded as a trigeminal symptom. At all events, trigeminal symptoms are mentioned in almost all of the cases, such as severe facial pains, facial paræsthesiæ, and

falling out of the teeth. The flow of saliva, the sialorrhœa, occurs usually in the form of attacks, which last a few days and during which great quantities of saliva may be discharged (about 500 gm. in twenty-four hours). The flow of tears, the epiphora of the tabetic, which is occasionally met with is to be regarded in the same way as the sialorrhœa.

Paralysis of the masticatory muscles is generally unilateral, and all the muscles of the affected side appear to be attacked. The muscles waste, a pit is felt in the situation of the temporal, and the jaw is deviated by the action of the normal pterygoid muscles of the other side.

Disturbances of the sense of taste are very rare in tabes. It is reported in exceptional cases that taste was absent upon the anterior part of one-half of the tongue (in the presence of other trigeminal symptoms) or that a patient was found with no sense of taste whatever, or with a morbidly altered sense. It is remarkable that the facial muscles are very rarely attacked in tabes. It is true that paralysis of the facial nerves sometimes occurs, but not every facial paralysis in tabes patients is of a tabetic nature. We may also doubt whether spasm of the face may not be due to some complication. I have twice seen convulsive tic in tabes patients in whom there was no trigeminal affection.

Both the disturbances of hearing and giddiness may be regarded as symptoms of involvement of the auditory nerve. Some authors, for example P. Marie, have found the so-called Menière's symptoms in a considerable number of their cases of tabes. These are subjective noises with attacks of giddiness. The noises were described as like the whistling of locomotives, or the sound of rushing or boiling water, or the sound of bells. The patients felt themselves impelled forward or to one side or as if they were being whirled about or as if sinking. Some had giddiness even in bed; they imagined that they were falling out, or were revolving with the bed. Deafness was generally present, but this was not dependent upon diseases of the nerve of hearing but upon affections of the middle ear not of a tabetic nature. According to the views of other authors with whom I agree, Ménière's disease is rare in tabes. Of course all giddiness is not to be referred to the semicircular canals. Its cause is often paralysis of the eye muscles. Many patients become dizzy if they look upwards, though nothing abnormal may be found in eye or ear. Some attacks of dizziness are probably slight epileptic attacks.

Disease of the auditory nerve is rare, but does occur. A unilateral or bilateral deafness is then present which gradually becomes complete. Irritative phenomena may or may not occur at the beginning.

Among symptoms of the vagus and accessory nerves the laryngeal crises and paralyses are the most important. Both conditions generally occur together, although the one is not dependent upon the other. The common laryngeal crises somewhat resemble an attack of whooping cough. A sensation of pricking, tickling, or choking in the throat generally precedes the attack, but the spasm may also develop quite suddenly. A long noisy inspiration is succeeded by short, quick coughs and finally a small quantity of mucus is expectorated. These attacks differ greatly in duration and severity. Contrasted with them are the still more severe attacks in which suffocation, loss of consciousness, and epileptic convulsions endanger the life of the patient. There is first a burning in the larynx followed by a feeling of suffocation, the patient can no longer stand, he struggles painfully to breathe and draws long, noisy inspirations. Soon even this becomes impossible, the patient loses consciousness, his pale face becomes cyanosed, and an epileptic paroxysm may ensue. Generally, however, the process does not go to this extreme; after a few minutes the spasm of the glottis relaxes and the patient recovers consciousness. It appears, however, that death may occur in these attacks. The ictus laryngeus has been described as a separate form; in this unconsciousness follows immediately the premonitory sensations in the larynx and the patient falls to the ground, so that the attack strongly suggests a common epileptic fit. The different forms of laryngeal attacks may occur in the same patient. He may suffer for years with slight attacks and the severer form may finally develop. The frequency of the attacks also varies greatly. Some patients have an attack only once in a year or two, others have them very frequently. These attacks may occur in series, so that all night long one attack of paroxysmal coughing may follow another. Sometimes no exciting cause can be detected, sometimes the attacks appear to be brought on by swallowing or by rapid bodily movements. Aside from the laryngeal paralyses examination discloses often an increased sensitiveness of the laryngeal mucous membrane; if the vocal cords are touched with a probe an attack is produced. In some cases laryngoscopic examination detects no abnormality.

As a rule, laryngeal paralyses are present together with the crises of the larynx, but may develop later than the latter, and may occur without them. By far the most common form of laryngeal paralysis is paralysis of the abductors. If this is unilateral it causes little trouble. If, as is usually the case, it is bilateral there is difficulty in breathing with rattling or gasping sounds as soon as the patients begin to move rapidly, ascend stairs, and the like. The voice remains clear, but the patients are easily fatigued by talking. Examination

shows approximation of the vocal cords and insufficient opening of the rima glottidis, which is often of an elliptical form. The abductor paralysis seems to predispose the patient to severe crises, since if a spasm occurs the glottis is easily completely closed. The paralysis next in frequency is unilateral paralysis of the vocal cords or recurrens paralysis. Here difficulties of respiration are not marked, but the voice becomes either harsh and metallic or quite devoid of resonance. The rarest form is complete bilateral paralysis of the vocal cords. Anæsthesia of the larynx is only exceptionally met with.

The laryngeal affections belong as a rule among the early symptoms of tabes. They are not very common. According to various statistics they may be expected in only about seven to eight per cent. of tabes cases, although the statements of the laryngologists show that tabetic paralyses of the larynx are the most frequent forms of laryngeal paralyses. In contrast with the paralyses of the ocular muscles, transitory paralyses are rare in the larynx. The laryngeal paralyses are generally persistent from their first appearance, although their degree may vary.

The pharyngeal crises described by Oppenheim are much more rare than the affections of the larynx. They consist in attacks in which violent movements of swallowing follow each other in quick succession attended by gurgling or cooing sounds. The attack lasts from a few minutes to half an hour. There may be twenty-four swallowing movements to the minute. It is said that deep pressure beside the upper portion of the larynx will generally stop these attacks.

Paralysis of one-half of the palate occurs sometimes, almost always in connection with other paralytic conditions which are to be referred to the last three cerebral nerves. Very rarely atrophy of the external muscles supplied by the spinal accessory, the sterno-cleido-mastoid and the trapezius, have been observed in tabes. This affection, like the peripheral muscular atrophy of tabes in general, which we shall describe below, is accompanied by the reaction of degeneration and may be either transitory or persistent. I once saw muscular atrophy confined to one trapezius which disappeared entirely in the course of a year.

Some authors regard the tachycardia of tabes as a vagus symptom. Acceleration of the pulse is not rare in tabes. In a certain proportion of the cases the affection is undoubtedly the tachycardia of exophthalmic goitre. Degeneration of the large arteries and of the heart may sometimes be the cause of the tachycardia. The angina pectoris which is occasionally observed is by some ascribed to the vagus as being a "cardiac crisis." I presume that in such cases some other complication was present.

It is doubtful how far the gastric crises are to be considered a vagus symptom. They will be considered below.

Finally the hypoglossus nerve also does not escape in tabes. Charcot, G. Ballet, and others have seen unilateral atrophy of the tongue. I have also seen it once in tabes. Hemiatrophia linguæ is generally first discovered during an examination, for it usually gives the patient no annoyance and interferes neither with eating nor with speaking. The protruded tongue arches towards the affected side. The atrophied half of the tongue forms a kind of appendage to the healthy side and is deeply furrowed. Since hemiatrophia linguæ is a rare affection, it at once suggests tabes, provided that peripheral disease of the hypoglossus nerve or a tumor or other affection of the base of the skull is not present. Lingual atrophy, as a rule, is found among the early symptoms. Exceptionally, bilateral atrophy of the tongue occurs; such a case was reported by Eisenlohr.

It is a singular fact that the symptoms of lesions of the cerebral nerves often appear in groups. Not only do different symptoms from the vagus and accessorius occur together, but also nuclei which do not lie near each other often become diseased at the same time, or in succession. Thus paralyses of the ocular muscles are always present with atrophy of the tongue, and together with trigeminal symptoms there are almost always symptoms from other cerebral nerves, etc. It appears, therefore, that there is, as a rule, a more or less extensive injury inflicted upon the region of the cerebral nerves if tabes once invades it at all. But it should be added that atrophy of other muscles is often associated with that of the muscles innervated by the cerebral nerves, for example, atrophy of the small muscles of the hand with atrophy of the tongue or of the muscles of mastication; further that bone and joint affections appear to be more frequent in patients with muscular atrophy than in other patients, and finally that gastric crises are not of rare occurrence in these cases. Thus there may be encountered a collection of the rarest symptoms of tabes in one individual. After perhaps a series of cases lacking in symptoms of unusual interest there comes one case in which such symptoms are present in superabundance. It is doubtful whether such phenomena depend upon a peculiarity of the tabes poison or upon an idiosyncrasy of the patient.

Besides the affections of the cerebral nerves, various phenomena are to be mentioned which are generally called the *cerebral symptoms* of tabes. These are attacks of migraine, epileptic attacks, apoplectiform attacks, and mental disturbances. It is probably more correct to designate these as symptoms of general paralysis in tabes patients. They correspond to an incomplete paralytic affection of the

cerebral cortex and there are easy transitions between cases with isolated paralytic symptoms and those with undoubted general paralysis.

Attacks of migraine may appear simultaneously with the signs of incipient tabes, not only attacks of the common migraine but also of ocular migraine. We must, of course, distinguish between migraine patients who become tabetic and tabes patients with attacks of migraine. In the former class the migraine is inherited and has existed since youth; the attacks often become more rare or cease altogether when tabes develops. If, on the other hand, the migraine is a part of the tabes, it first begins at a mature age. If a man of forty years has an attack of migraine for the first time we may think seriously of the possibility of tabes. Such tabetic or paralytic migraine attacks do not, however, appear to be very common.

Epileptic attacks are also rare in tabes. Perhaps some of the attacks of giddiness in tabes patients are of an epileptic nature. The previously mentioned epileptic conditions connected with the laryngeal crises are of course not to be considered here. On the other hand, it is possible that epilepsy may occasionally occur, not subordinated to tabes but simultaneously with it as a coördinated affection. There is a parasyphilitic epilepsy (Fournier), that is, an epilepsy which bears the same relation to syphilis as tabes does.

Somewhat more frequent than epileptic are apoplectiform attacks, suddenly developing and as suddenly disappearing hemiplegia, monoplegia, aphasia, etc. These much resemble the paralytic attacks, and as a rule other paralytic symptoms are present in the cases in which they occur. Yet it is said that sometimes the apoplectiform conditions are the only paralytic symptoms present. Of course cerebral focal lesions which are due to arteriosclerosis are occasionally observed in the tabetic. If the cerebral paralyses are permanent, the existence of such foci is probable.

Well-marked psychical changes no one would regard as symptomatic of tabes, yet lesser changes which correspond to a light paralytic affection of the cortex, as has been repeatedly proved, seem to be not very rare. There is then a slight mental weakness with some euphoria. It has been repeatedly observed that many tabes patients endure their severe affliction with remarkable cheerfulness and hopefulness, and it is known that they readily entertain all kinds of deceptive notions. In some a distinct loss of memory is also observed. It is often naturally impossible to determine with any certainty whether the mental powers of the patients are at all impaired.

If true insanity develops in tabes patients, its form in the great majority of cases is general paralysis, but of course all of the other

forms of mental derangement may occur in tabes, to wit, paranoia, intermittent insanity, hypochondriacal and hysterical states, etc. The distinction will generally not be difficult, since in the endogenous psychoses abnormal mental states have generally been present before the appearance of tabes, and the clinical picture itself generally admits of no doubt. Of course, complicated conditions occur—endogenous aberrations plus alcoholism, plus tabes, or perhaps morphinism plus traumatic hysteria, plus tabes, conditions in which careful inquiry into the history and continued observation are necessary in order to reach a decision. At all events, the so-called "tabes psychosis" does not exist. It has been supposed that the pains, the paræsthesiæ, etc., might lead to a disturbed psychical state in which the perceptions become distorted, the pains being regarded as produced by invisible enemies and the like. But such assumptions are purely arbitrary; as a rule in the tabes psychosis the condition is general paralysis, or, exceptionally, tabes with paranoia.

Besides the symptoms already discussed the most important signs of tabes consist in two groups of phenomena; first, affections of the viscera which manifest themselves partly as lancinating pains in the thoracic and abdominal organs, and partly are probably to be explained as the results of anæsthesia of these organs; and second, changes in the bones, the muscles, and the skin with its appendages, changes which are generally comprehended under the term trophic disturbance.

Visceral crises consist in attacks of pain in the stomach, the intestine, the bladder, or other organs. Secretion generally accompanies these pains and sometimes the attacks are purely secretory, without pain.

By far the most important and the most common are the gastric crises. Delamare first called attention to them and to Charcot we owe the first correct description of them. Pain generally occurs quite unexpectedly in the region of the stomach—"cramps in the stomach." Sometimes the pain is described as a contraction, and sometimes the patients speak of electrical shocks which pass from the stomach to the spine and occasionally to the region between the shoulder blades. Girdle pain also occurs. Emesis soon appears, and returns at short intervals, somewhat as in seasickness. The food which happens to be in the stomach is first vomited, then follows watery mucus, which is soon mixed with bile. Blood is not infrequently vomited also. It is impossible to take nourishment, for everything, even a swallow of water, is vomited immediately. The pains and the vomiting continue for hours or days, even weeks. The average duration of a gastric crisis is several days. The pains are often very

severe, so that the patients cry out, are "doubled up" with them, writhe, and assume the most extraordinary positions, and may even faint. Hardly less severe sufferings come from the continuous vomiting and retching, the thirst, and the loss of sleep, so that it is easily comprehended why the patients are greatly exhausted and depressed after a crisis of some duration. It is remarkable that the stomach appears to be normal immediately after the termination of a crisis. One of my patients, after he had eaten absolutely nothing and had vomited incessantly for days, was accustomed to call for a glass of Bavarian beer, which he greatly relished. After this he could eat anything. There are crises which last only half an hour or an hour, and, on the other hand, states lasting for weeks have been observed in which transient improvement is always followed by renewed pain and vomiting. Of late years, many investigations have been undertaken to ascertain the chemical constituents of the vomited matters, with varying results. Great acidity has been found only a few times; more frequently the amount of acid is small. In all probability the chemical condition of the contents of the stomach is of quite subordinate importance; it varies with the state of the patient. The amount of blood vomited is rarely large. As a rule it is produced probably by mechanical injuries of the mucous membrane during retching. There may, however, be hemorrhages which are analogous to the cutaneous hemorrhages which sometimes accompany the lancinating pains.

The incomplete gastric crises in which there is either pain alone or only retching and vomiting are rather uncommon. In the former case the condition resembles an attack of gastric colic and may easily fail to be recognized, in the latter the crisis is very similar to seasickness, and diagnostic errors may be committed especially if hyperacidity is present.

Intestinal crises are very rare. By this term is denoted attacks of diarrhœa which, in contrast with the gastric crises, are only rarely attended with pain. The watery diarrhœa begins suddenly and apparently without cause. There are four, six, eight, or more evacuations a day. The affection continues for days, weeks, or months, and then suddenly disappears. This diarrhœa much resembles that of morbus Basedowii and it may well be that in some cases of tabetic intestinal crises an unrecognized Basedow's disease has been present.

Occasionally when the expression "intestinal crisis" is used, the rectal pains which also occur in paroxysms are meant. The patients say that it seems as if a wedge or a red-hot iron were driven into the bowel. Sometimes also very annoying tenesmus occurs, which may be attended by the evacuation of bloody mucus.

As a rule the intestine is sluggish in tabes patients. They suffer from constipation and are obliged to take laxatives. True intestinal incontinence is very rare, even in the late stages of the disease. Yet not very infrequently small quantities of fæces pass off unnoticed, so that the patients complain that notwithstanding their best efforts they are unable to prevent soiling their clothing. With these cases those should not be confounded in which the patients are obliged to strain while urinating and so occasionally force out a part of the contents of the rectum.

Gastric and intestinal crises belong among the early symptoms of tabes. They may appear as the first signs of the disease and may continue for years before other symptoms noticeable by the patient manifest themselves. Sometimes they become slight and infrequent or cease entirely, when the usual symptoms of tabes develop. The statements as to their frequency do not agree, but we may assume that they occur in from three to five per cent. of the cases. We have already spoken of the relatively frequent occurrence in combination of disturbances of the larynx, gastric attacks, and bone disease. The great majority of tabes patients, although constipated, have a good digestion.

Vesical crises are sometimes encountered. They consist in violent pains which, starting from the hypogastric region, radiate along the urethra and extend to the perineum and the inner side of the thighs. The patients state that it seems as if water were rushing through the urethra or as if molten lead were running through it. In spite of a painful impulse to urinate, they are unable to do so. The attack lasts several hours or even a day, and often ends with the passage of blood. It may return at intervals of weeks and may appear in connection with the lancinating pains in the legs. It is of course necessary to be cautious in making the diagnosis. I believed once that one of my patients had a vesical crisis, but it finally appeared that there was a small calculus in the bladder, after the passage of which the attacks ceased. Yet the authors are positive that there were no calculi in their cases. In Raynaud's first case nothing abnormal was found in the bladder.

Renal crises have also been described, that is, attacks which resemble those of renal colic. The known facts are, however, so scanty that it is well to reserve an expression of opinion regarding them.

Although the process of urination in tabes is often affected, yet the quantity and constitution of the urine are rarely abnormal. Polyuria has occasionally been seen and in a number of cases glycosuria. In tabetic glycosuria the amount of sugar is always small—one-half, one, at most two per cent. Larger amounts are probably found only

when the tabes is complicated by diabetes mellitus. The glycosuria is generally referred to an affection of the medulla oblongata. As a matter of fact, bulbar symptoms have repeatedly been found present with it. The so-called alimentary glycosuria is also said to be sometimes met with in tabes. There is probably always some complication, if albuminuria is present.

In male patients impotentia coeundi generally develops sooner or later. It may appear at a very early or at a relatively late stage. In rare cases the function suddenly disappears forever; as a rule, the desire gradually diminishes and erections become more and more weak and infrequent. The potentia generandi is probably often preserved unless a complication (epididymitis, sarcocele) is present. But finally an atrophy of the testicles may develop.

Leimbach found a diminution or abolition of the sexual appetite in 58.25 per cent. of the patients. In 15.54 per cent. this was the first symptom. It is probable that the anæsthesia plays an important part in the bladder affections and in the impotence. But anæsthesia of the external parts is not necessarily present. In exceptional cases, priapism and increased sexual desire are reported in incipient tabes.

Anæsthesia of the vulva and of the vagina is sometimes found in women. Pleasure in the sexual act is then usually absent, but it is sometimes wanting when anæsthesia cannot be detected. Menstruation is as a rule unaffected, but occasionally ceases.

The crises of the clitoris constitute a singular symptom. The patients have the sensations and the secretion that accompany coitus. The attacks may occur in the daytime as well as at night. In the latter case they are sometimes accompanied by dreams, and then resemble what has been described as "pollutions in the female." The disagreeable feature in these clitoris crises consists in the fact that they sometimes precede an attack of lancinating pains. This symptom is rare; I found it only once among fifty female patients.

Among the most interesting features of tabes are the diseases of the osseous system which manifest themselves chiefly as fractures and as the so-called *arthropathies*. The tabetic arthropathies were first described by Charcot. His masterly description has been subjected to much criticism, but is gaining more and more universal acceptance. The tabetic joint affections are often, especially in England and America, called "Charcot's disease," a term which is not to be recommended, for the French sometimes call amyotrophic lateral sclerosis Charcot's disease. The joint affection develops without apparent cause, and as a rule in the early stage of tabes, often before the ataxia. It generally appears suddenly without any premonitory

symptoms, manifesting itself by a large serous effusion into the joint and a doughy swelling of the surrounding parts. Fever and pain are generally entirely absent. Either the swelling disappears after a time and the parts again become normal (benign form), or severe lesions are left within the joint, and we find crepitation, dislocations in consequence of the wearing away of the surfaces of the bones, or other luxations (severe form). The knee is most frequently attacked, then the hip joint, the elbow, and the wrist; more rarely the joints of the foot, of the vertebræ, and of the fingers.

On post-mortem examination there are almost always found atrophy and proliferation, the former as a rule greatly preponderating. If the process has not advanced very far a part of the articular cartilage is still preserved. This appears eroded. In the bone is found sclerosis together with spongy atrophy. Sometimes a ring of osteophytes surrounds the articular surface. The joint capsule is dilated and thickened and often studded with excrescences. The cavity of the joint contains a viscid fluid, and loose bodies in the joints are often present. Suppuration is extremely rare, if present being due to a secondary infection. In the later stages the joint surfaces together with the cartilage have disappeared. The entire articular extremities may be worn away, so that, for example, the head and neck of the femur as well as the acetabulum have disappeared. In some cases proliferative processes are more marked, so that thick layers of newly formed bony tissue surround the joint. On chemical examination the bone is found to be poor in mineral constituents, especially the phosphates, and rich in fat. Microscopically there are found a rarefying osteitis and irregular dilatation of the Haversian canals, which are filled with fat. But the anatomical examination furnishes a picture of the termination of the process only. Most truly characteristic is its course—the sudden and painless development of an enormous swelling and the often rapid destruction of the joint, which in a short time may lead to subluxations or luxations.

In view of this clinical picture there can be no question that tabetic arthropathy is not the same as arthritis deformans. It is a quite distinct affection, such destruction of joints occurring elsewhere only in syringomyelia. The origin of the articular affection is to be explained by supposing that the first change is a morbid alteration in the bone. Accordingly as the diaphysis or the epiphysis is chiefly affected, there next results either the fractures which are to be described further on, or the arthropathy. In the latter it often appears that the process is initiated by the breaking off of small portions of the articular ends. The large swelling which is par-

ticularly marked in case the knee or the shoulder is affected is sometimes caused by the distended joint capsule, but sometimes it is diffuse, the lower part of the thigh and the greater part of the leg, for example, being markedly œdematous. In this case there is probably a rent in the capsule through which the articular effusion becomes diffused. If the joints of the instep are affected a singular deformity is produced, the so-called tabetic foot. A hard boss projects upon the dorsum and in the middle of the sole, the foot becomes glossy and is shortened, the parts of the arch of the foot being, so to speak, driven into one another. The movements of the foot are painless, but are attended with crepitation. In the rare cases of tabetic disease of the lumbar vertebræ spondylolisthesis may result. The arthropathy is often bilateral, both knees, both hip joints, or both feet being affected.

Since the entire osseous system is manifestly involved in tabes, if the disease of the bones is marked there are numerous arthropathies and fractures. A female patient of Charcot's had luxation of the tibia backwards in the left knee with marked wearing away of both articular ends and almost complete disappearance of the patella. Both hip joints were diseased, as shown by loud cracking and crepitation, there was luxation of the right femur backwards, the left humerus was luxated inwards, and its head and the glenoid cavity had disappeared; in the right maxillary joint there was loud cracking on movement. Upon post-mortem examination of this patient fractures of the malar bones and other lesions of the osseous system were discovered which had escaped clinical observation. Kredel has collected a large number of cases of tabetic arthropathy. According to him, the knee joint was affected 104 times, the hip joint 56 times, the shoulder joint 36 times, the elbow joint 15 times, the ankle joint 25 times, the toe joints 10 times, the finger joints 8 times, the maxillary joint twice; the "tabetic foot" was present in 16 cases.

Fractures of the diaphyses in the tabetic are more rare than the joint affections. It is characteristic of them that they are produced by quite trivial causes, hence the name "spontaneous fractures." The patient, for example, fractures his thigh while pulling on his boots; even turning in bed may cause a fracture. The fracture is almost always painless, and with proper treatment generally knits well. If the fragments are not held immovable, unsightly masses of callus may be formed. It is especially to be noted that the spontaneous fracture may appear among the first signs of tabes. If a fracture of the thigh or the leg, produced without adequate cause, is found in a man in whom the possibility of tabes cannot be excluded, that disease should always be suspected. Other igns of the disease (reflex irido-

plegia, etc.) will generally be already present, but it would seem that spontaneous fractures may occasionally precede all the other signs. Kredel collected 73 cases of spontaneous fractures in tabes; the thigh was implicated 32 times (among these 9 fractures of the neck of the femur), the leg 19 times, the forearm 6 times, the arm 4 times, the clavicle twice, the pelvis 3 times, the scapula twice, and the lower jaw and the radius each once. Multiple fractures occurred in 16 cases, and in 16 cases fractures and arthropathies were present at the same time.

As to the question whether the bone affections in tabes depend upon definite lesions of the nervous system, and what these lesions are, opinions differ. Charcot was at first inclined to assume an affection of the anterior cornua. There is now no doubt that his view is incorrect. Others supposed the cause to be lesions of the peripheral nerves, especially of the nerves which penetrate the bones—a view the reasons for which are difficult to understand. Recently Marinesco and others have regarded the anæsthesia as the causal affection. In health the nutrition, that is, the blood supply of the bones and of the articular surfaces, depends upon centripetal stimulation. If this is wanting as in tabetic anæsthesia, the nutrition suffers, because the reflex regulation of the blood supply is no longer active. It may also be supposed that the poison of tabes may injure the bones directly under certain conditions, that consequently the bone affection is coördinate with that of the nerves.

The *ligaments* may also be destroyed in arthropathy. The tendinous tissue probably at times suffers a morbid change in tabes analogous to that of the osseous structures. The occasional ruptures of tendons point to this. By a sudden movement the Achilles tendon may be ruptured or the quadriceps tendon may be torn from the patella. These injuries are painless, but the muscle which is separated from its point of attachment contracts, and union does not generally take place.

Of the *diseases of the skin* in tabes the perforating ulcer is closely related to the affections of the bones. Not only is it found especially in the cases in which arthropathies and fractures are also present, but it often seems to depend upon an affection of the bones. By malum perforans is meant an ulcer which perforates the skin and at the base of which carious bone is often to be felt. It manifests itself almost always as small cavities in the sole of the foot or on the dorsum of the toes, looking as if they had been punched out, which secrete a thin, ill-smelling pus, are insensitive and heal with difficulty. It is manifestly possible not only that a neglected injury of insensitive parts of the foot might lead to a deep ulcer which finally reaches the

bone or penetrates into a joint, but also that dead bone may originate a fistula which appears as malum perforans. The suppuration of a bursa may also be the first step in the process. The favorite location of the ulcers is the toes, and of these especially the great toe. Several ulcers often occur simultaneously or in succession.

Other changes in the skin are very rare. Erythema, vitiligo, and a condition resembling ichthyosis have been described as signs of tabes. The last-named condition consists in a reddened, dry, and thickened skin, covered abundantly with epidermic scales. Occasionally obstinate chronic eczema is found upon the hands and feet. The cutaneous hemorrhages and the herpetic eruptions which occasionally accompany the lancinating pains have already been mentioned. Atrophy of the skin also occurs. The skin is then either wrinkled and thin, as if too large, or thin, glossy, and closely adherent. More frequent are changes in the sweat secretion, hyperhidrosis, or more commonly anhidrosis. It is well known that patients who have formerly had sweating feet, losing this excessive secretion at the beginning of tabes, are apt to regard this loss as a cause of the disease. Anhidrosis (or hyperhidrosis) of the lower half of the body, of one leg, of one arm, or of one-half of the face is also met with.

Finally *affections of the nails* should be mentioned. The nails, especially those of the great toes, are sometimes peculiarly thickened, uneven, and discolored. In some cases they fall off, and this process is sometimes preceded by violent pains, and there may be repeated hemorrhages under the nails. This symptom also occasionally occurs in connection with perforating ulcers and with atrophies.

Muscular atrophy in tabes appears in various forms. There may be an isolated affection of individual muscles, which disappears or becomes stationary, or, as in the so-called neurotic atrophy, many muscles may be affected with atrophy simultaneously. The atrophy generally begins insidiously. The extremities are first attacked, and only exceptionally does the process begin elsewhere than in the hands and feet. In nineteen cases reported by Déjerine the feet were first involved nine times, the hands seven times, the shoulder muscles and the flexors of the forearm only once each. If the atrophy attacks the legs, the small foot muscles first become atrophied and the foot soon becomes strangely distorted. The interosseus spaces are sunken, the balls of the toes lose their roundness, the terminal phalanges are bent backwards, and the other phalanges are flexed towards the plantar surface, but the great toe is generally flexed in both joints. If the patient attempts to raise the foot, a true "claw foot" results. Sometimes the muscles and ligaments shrink and fix the foot in an abnormal position. At a later time the muscles of the leg are also implicated,

the peroneal group especially, the tibialis anticus being occasionally spared for some time. Then a pes equinovarus results and the "claw" position of the toes changes to one of complete flexion. Gradually the foot becomes fixed by the shrinking of the calf muscles and of the peroneus longus (clubfoot of the tabetic). In the hands the thenar eminence is first attacked, then follow the other small hand muscles and occasionally also the muscles of the forearm. The atrophy is not as a rule to be distinctly recognized above the knee and elbow, but, as post-mortem investigation shows, the muscles of the thigh and arm do not always escape entirely. The atrophy is almost always approximately symmetrical. Fibrillary twitchings are said not to occur. The response to both mechanical and electrical stimuli is diminished, and sometimes it is possible to detect the reaction of degeneration. Progressive tabetic muscular atrophy is a rare affection, and occurs almost solely in the later years of the disease. Autopsy shows degeneration of the muscles and of the muscular and cutaneous nerves. The degeneration is also present in the mixed nerves, but decreases as they ascend, the anterior roots, as a rule, not being materially altered, and the anterior cornua being quite normal.

In the atrophy which is confined to single groups of muscles (aside from the muscles innervated by cranial nerves which have already been considered) the muscles affected are some of the small hand muscles, or the peroneal muscles, occasionally the deltoid of one side, etc. Such circumscribed atrophy may occur in incipient tabes and not rarely it may terminate in recovery.

Finally those usually transitory *paralyses* should be mentioned which are called nerve paralyses because the region of a definite nerve is attacked. Peroneal and radial paralyses are especially to be considered. In the latter the common cause, *i.e.*, pressure on the nerve during sleep, can often be made out but not invariably. In one case, for example, the paralysis occurred while the patient was turning over the leaves of a book. In some cases pressure appears to have been one of the causes, but such slight pressure that it could not have caused paralysis in a healthy individual. Déjerine believes that the tabetic radial paralyses are characterized by a special electrical reaction, viz., that the nerve has lost its excitability not simply above the point of pressure, as is usually the case, but throughout its extent, while the faradic excitability of the muscles is preserved. I have not been able to confirm these statements, finding merely diminution of the excitability.

To explain the transient tabetic paralyses we must suppose that the whole nervous system is injuriously affected in tabes so that rela-

tively slight injuries may produce disturbance of function or nerve destruction.

Many phenomena point to the existence of slight disturbances of the motor portions of the nervous system also, which have as yet not been mentioned. As, for example, the sudden giving way of the legs; the patients feel suddenly that their strength is leaving them, and are obliged to lay hold of something for a support or to fall down. A part of the great weariness and objectively demonstrable muscular weakness of the tabetic is certainly not to be explained by the anæsthesia alone. Attacks of fatigue have been described, in which the patients feel an exhaustion as if after mountain climbing or other arduous exertion.

Old tabes patients, even when no true paralysis and no muscular atrophy are present, often feel so weak that they can hardly move their limbs. In such cases the autopsy reveals, as a rule, a degeneration of the lateral tracts, the formerly slight and not demonstrable affection of the motor paths having led finally to visible decay.

The foregoing description of the signs of tabes is reasonably complete. But one group of disturbances upon which much study has been expended has not yet been mentioned. I refer to diseases of the *heart* in tabes, cases with cardiac lesions being found with relative frequency in that disease. According to recent statistics (Nordmann) 1 out of 11 tabes patients has disease of the heart, but according to Leimbach there were only 7 patients with heart disease in 400 cases of tabes. Of 130 cases of heart disease in tabes there were 51 with aortic lesions (38 insufficiency, 7 stenosis, 6 insufficiency and stenosis), 4 with aneurysms, 10 with aortic and mitral lesions, and 33 with mitral lesions alone. In the remaining cases the affection appears to have been chiefly myocarditis. Symptoms due to or suggesting angina pectoris were mentioned in 23 cases. On closer examination it appears that the mitral lesions in tabes have the same causes as in other affections (chiefly polyarthritis). Although there has been no lack of more or less ingenious argument to prove that the diseases of the aorta and of its valves are due to tabes, or even that the latter depends upon the former, it is highly probable that the two morbid conditions are coördinate; in other words, that the aortic lesions as well as the tabes are metasyphilis, and that they consequently represent a complication of tabes like the contracted kidney.

Course.

The course of tabes is generally divided into three stages; the first or the pre-ataxic stage, the ataxic stage, and the paraplegic stage.

The duration of the first stage may vary from one or two to twenty

years or more; indeed many tabes patients die before they pass beyond it. We may say either that the duration of the first stage is unlimited or that there are incomplete, abortive forms of tabes. I consider the latter statement the more correct, but it is always possible to say that if the patient had lived longer, the tabes would have become more fully developed. This objection is admissible even if the disease has been at a standstill for a long time, for even then it may, so to speak, become revivified and advance progressively. The most simple form of the first stage is that in which the patient has pains from time to time—"his old rheumatism"—and otherwise regards himself as healthy. The physician generally finds reflex iridoplegia and absence of the knee phenomenon. Sooner or later slight vesical disturbances appear. In other cases a paralysis of the ocular muscles occurs from time to time. The patients are generally constipated and their sexual powers are slight.

To this simple relatively benign tabes can be contrasted the more or less malignant form in which from the first, or at least during the first stage, the rarer symptoms appear in larger or smaller groups, such as multiple affections of the cerebral nerves, laryngeal symptoms, gastric crises, arthropathies, and fractures. All these symptoms and others may be present in the first years, and each of them is of bad omen. Tabes with atrophy of the optic nerve may be regarded as a form of subordinate importance. The patients are severely afflicted by their blindness, but in other respects do not suffer greatly.

A simple tabes may become malignant if the pains become frequent and severe, or if the vesical disturbances increase rapidly.

The second stage, that of ataxia, is generally introduced by Romberg's symptom. The patients become somewhat uncertain in the dark, it is more difficult for them to descend poorly lighted stairs. The anæsthesia upon which these phenomena depend may remain unchanged for a considerable time, but it does finally increase, rapidly or slowly, and then true ataxia makes its appearance. As has already been mentioned, the rarer acute or subacute ataxia of tabes is to be distinguished from the common slowly increasing ataxia.

Of the patients who reach the second stage, a considerable number die before entering the third stage from an accidental complication, from a metasyphilitic disease coördinate with the tabes, such as renal or cardiac disease, from general paralysis superadded to the tabes, or finally from a pyelonephritis resulting from paralysis of the bladder. If the patient continues to live, the ataxic stage generally lasts several years. The cases in which the patients are bedridden after about a year's duration of this stage are rather rare.

Often, not always, the pains diminish in the ataxic stage or cease entirely. In other respects the condition becomes increasingly more complicated, since, on the one hand, the symptoms already present develop more fully, and, on the other, new ones make their appearance in greater or smaller numbers. The anæsthesia differs greatly in different cases. Sometimes anæsthesia of the skin is almost completely absent and that of the deeper parts is apparently slight in spite of marked ataxia. In other cases the anæsthesia is conspicuous, of wide extent, and abounding in individual variations. Most clinical investigations of anæsthesia have been made upon such patients and most of the forms of anæsthesia have been studied in their cases. In the ataxic stage the formerly transient paralyses of the ocular muscles often become permanent. The gastric and other crises may now cease, but they are often found in connection with the ataxia. New symptoms of various kinds may be added at any time to those already present. The multiplicity of the combinations is so great that no description can be adequate.

If we speak of the third as the paraplegic stage, we do not mean that true paraplegia is always present in it, although this does frequently occur. It may be early and transient, ushering in perhaps the subacute ataxia, or it may develop slowly at a late period as a persistent symptom corresponding to a degeneration of the lateral tracts of the spinal cord. But generally in tabes there is never true paraplegia. The patients are confined to their beds only by ataxia and weakness, and this stage might therefore be called more correctly the bedridden stage. In many patients articular affections, fractures which have resulted in pseudoarthrosis and muscular atrophy are additional causes of the inability to move about. Death is produced sometimes by infection of the urinary passages, sometimes by decubitus, which is to be explained in part by depressed nutrition of the tissues in general, in part by the action of decomposed secretions upon the insensitive skin, sometimes by the development of paralysis, and sometimes by the complications already mentioned. The third stage is shorter than the second, and seldom lasts more than a few years.

On the whole, then, the course of tabes is a slow change for the worse, and it deserves therefore the adjective progressive. In the individual cases, however, we find that at any time (excepting perhaps in the third stage) the progress of the disease may be checked for a long, occasionally we might say an unlimited period, and not only that, but there are ameliorations which may be very marked. Such ameliorations, which are in part very difficult to understand, have great practical importance. In rare cases all the subjective symptoms are said to have disappeared, although the autopsy showed unmistakable ta-

betic changes. Rather frequently the symptoms disappear, for a time at least, to such an extent that the patients who are as a rule very hopeful think they are cured, and regard themselves as well for one or two years or even a longer period.

DIAGNOSIS.

The diagnosis of tabes is, as a rule, very easy. The mistakes which are often made even to-day are due, not so much to the difficulties of the problem as to careless and hasty examination. It is only necessary to know what the signs of tabes are, and when one of these signs is present to examine with reference to this disease. If the physician has tabes in mind, and if he makes his examination systematically in order to discover the chief signs, he will seldom fail to recognize the disease. If, for example, a patient with bladder disturbances, or one with pain in the stomach, or one with spasmodic cough presents himself, and no tangible changes in the organs in question can be detected, one should bear in mind the possibility of the existence of tabes. He who does not think of tabes first of all in a case of ocular paralysis, or of arthropathy of the tabetic kind, or of mal perforant, or of bilateral paræsthesiæ in the regions supplied by the ulnar nerve, or of darting pains in the legs, is simply ignorant.

If any sign is present which may belong to tabes, the pupils are to be examined first of all. If reflex iridoplegia is found the question is practically decided. If we exclude the exceedingly rare lesions (especially tumors) in the vicinity of the corpora quadrigemina, reflex iridoplegia is a symptom which by itself suffices for the diagnosis of tabes. From a practical standpoint reflex iridoplegia is equivalent to tabes.

Secondly the knee phenomenon is to be investigated. It is absent in tabes, as a rule, but may be present and even temporarily exaggerated. It may be absent in other diseases, such as neuritis, myelitis of the lumbar cord, syringomyelia, cerebellar tumors, etc. Consequently the condition of the knee phenomenon is far less important than that of the pupils, and its absence can justify the diagnosis of tabes only when it occurs in combination with other symptoms.

In the third place are the lancinating pains. They are very characteristic. As they occur in tabes they are rarely found elsewhere, really only in some forms of multiple neuritis. Of course every pain in the legs is not to be considered; those which are diagnostic are the darting pains which appear now here, now there, are located chiefly in the muscles, return in paroxysms, and are associated with supersensitiveness of the skin.

The vesical disturbances are fourth in importance. If the knee

phenomenon is absent and lancinating pains are found to exist aside from tabes, no condition really requires consideration except polyneuritis. But if bladder disturbances are also present, the latter may probably be excluded. Diabetes might be thought of and the urine should be examined in every case. If sugar is present, the condition of the pupils will be decisive, for reflex iridoplegia is not one of the symptoms of diabetes. If the pupils are normal, the diagnosis between tabes and diabetes must depend upon the whole aspect of the case; it is impossible to decide the matter off-hand.

Reflex iridoplegia, vesical disturbances, lancinating pains, and absence of the patellar reflex constitute the basis of tabes. If they are wanting, only a probable diagnosis can be made. Yet some signs may precede these. Among the first may be atrophy of the optic nerve, paralysis of the eye muscles, gastric crises, ataxia and sensory disturbances, fragility of the bones, and some other rarer symptoms.

Atrophy of the optic nerve, *i.e.*, the whitish discoloration of the papilla which begins in its external half and slowly progresses, with thinning of the vessels but no other changes, is to a certain extent pathognomonic, and even if it appears alone, makes tabes very probable.

The majority of all ocular paralyses are tabetic. If an isolated paralysis of an ocular muscle appears in a man who has had syphilis some years previously, tabes is more probable than any other disease. Paralyses of the eye muscles from tertiary syphilis are almost always accompanied by violent pains. Those of incipient tabes, on the other hand, are painless and generally transient. On account of the latter fact they appear to yield to antisyphilitic treatment, and many physicians for that reason place them among the syphilitic paralyses of the eye muscles. Especially characteristic of tabes are the paralyses which correspond to a slight degeneration of the whole nuclear region, namely, bilateral abducens paresis and enfeeblement of all motions of the eye, which reveals itself by nystagmus-like twitchings in the extreme positions of the eye. Ophthalmoplegia interna, or isolated loss of accommodation in persons liable to tabes, makes the diagnosis very probable.

In isolated laryngeal paralyses local causes, especially pressure upon the recurrent nerve, are of course to be excluded. If this is done and if there is nothing to exclude tabes, this disease must be first thought of.

Gastric crises occur almost exclusively in tabes. If one has once seen a characteristic attack, there is no longer any danger of confounding the gastric crisis with a colic from gall-stones or a so-called

cramp of the stomach or anything else. Aside from tabes, gastric crises occur only in those extremely rare cases in which they are the sole malady, occurring from early youth; the relation of such cases to tabes is as yet by no means clear. There are, it is true, gastric crises which present nothing characteristic, incomplete attacks, from which alone the diagnosis cannot be made.

Ataxia very rarely occurs as the first symptom. I recall but one case in which, besides incipient atrophy of the optic nerve, the ataxia, with the disturbances of sensibility upon which it depends, was the first symptom without reflex iridoplegia, without loss of the knee phenomenon, and without pains and bladder disturbances. In such cases the diagnosis is of course uncertain.

The bone affections are also quite characteristic. Fragility of the bones may manifest itself at a very early period, and may probably be the first symptom. In every fracture without sufficient external cause tabes should be thought of. The arthropathies too may lead to a diagnosis without the necessity of other signs. If a huge joint swelling, accompanied by the crackings of the joint, develops suddenly without cause, tabes should be the first thought. Only he who has no individual experience, who perhaps is familiar only with anatomical dissections, can deny the *sui generis* character of tabetic articular affections. The same is true of the tabetic shedding of the teeth, of perforating ulcer, and of many other symptoms.

It is hardly possible, if any care is taken, to fail to recognize well-developed tabes. But it may be difficult to decide whether tabes alone or some other affection also is present, or whether besides some other affection tabes may not also exist. The distinction between tabes and progressive paralysis is first to be considered. As I have already explained, I am of the opinion that there is really but one tabes paralysis, but that the disease sometimes especially affects the brain (progressive paralysis, or tabes of the brain), sometimes the peripheral and spinal paths (tabes, or progressive paralysis of the spinal cord), sometimes both at once (tabes paralysis *sensu strenuo*). In practice, however, it is very esential to be able to distinguish the two forms, which in essence are closely related but clinically are different. If progressive paralysis undoubtedly exists, it is practically of slight importance to decide whether true tabetic symptoms are also present. On the other hand, it may be very important to decide at an early period whether paralytic symptoms are presenting themselves together with those of tabes.

In the beginning it is frequently not possible to decide what the further course will be. If, for example, a patient has only abducens paralysis and reflex iridoplegia, new symptoms which appear later

may be either paralytic or tabetic in character. In such cases the slightest signs of paralysis, for example quivering of the tongue or twitching at the corners of the mouth, are of great importance. But, even if such symptoms are found, it is well not to express too positive an opinion to the patient's friends. For there are cases of tabes with, so to speak, abortive paralysis, that is, the tabes may continue to develop as usual, while the paralysis does not increase beyond the first symptoms, or at least is of extremely slow growth. In the course of an undoubted tabes paralytic symptoms may also appear, but no well-marked progressive paralysis results. Thus there occur in the tabetic apoplectiform or epileptiform attacks, which are to be regarded as of the nature of paralysis, or a slight mental weakness appears which does not increase, or trembling of the hands manifests itself. If in incipient paralysis tabetic symptoms (absence of the knee phenomenon, vesical disturbances, lancinating pains, etc.) are found, their presence has a prognostic importance, inasmuch as, in general, paralysis with tabetic symptom runs a slower course than the form without such symptoms, in which latter symptoms of implication of the lateral tracts generally appear, such as increase of the reflexes and spasms. Reflex iridoplegia may occur in both forms of progressive paralysis.

Tabes may be found in connection with tertiary syphilis and it is possible that in the presence of the signs of lues cerebrospinalis those of tabes may be overlooked and *vice versa*. For example, the symptoms of meningomyelitis dorsalis and also reflex iridoplegia might be present; or, together with tabes, a multiple cranial-nerve paralysis depending upon exudative meningitis at the base of the brain might develop. If some symptoms in the tabetic appear to point to tertiary syphilis of the nervous system, antisyphilitic treatment is of course indicated. If in lues cerebrospinalis some signs of tabes are found, the hopes based upon the antisyphilitic treatment disappear.

Tabes occurs in connection with exophthalmic goitre. Fully developed Basedow's disease is rare with tabes, more frequently there are some signs of that disease, for example, slight goitre, accelerated pulse, and some exophthalmus. Perhaps the tachycardia which some count among the symptoms of tabes is to be regarded rather as a sign of exophthalmic goitre. The tabes patients may have an old, so to say idiopathic goitre, or in other cases the connection may be that a chronic syphilitic thyroiditis is the cause of this symptom.

Much has been written as to the connection of tabes with hysteria. Since both diseases are common it is not strange that they should sometimes occur together. Either there is no causal connection be-

tween them, a hysterical individual becoming syphilitic and in consequence of that tabetic, or the tabes may be the exciting cause, the tabetic symptoms, especially the pains, awakening the slumbering tendency to hysteria. Serious diagnostic difficulties can hardly arise from the union of tabetic and hysterical symptoms. We may indeed doubt whether this or that pain, paræsthesia, or anæsthesia, is to be referred to tabes or to hysteria; hysteria may caricature ataxia, but this obscures the diagnosis in respect to conditions of subsidiary importance only. It is most probable that slightly marked hysteria, existing side by side with tabes, will be overlooked and some therapeutic triumphs may depend upon such an error. Only the ignorant can confound tabes with hysteria. An hysterical individual brought into contact with the tabetic in the hospital or elsewhere might become familiar with their pains and disturbances of sensibility and motility and instinctively imitate them, but even then the chief symptoms of tabes are wanting—the reflex iridoplegia, the loss of the knee phenomenon, in short all the symptoms which are not producible by suggestion. On the other hand, other signs of hysteria will certainly be present which will guide the examiner in the right way. It is scarcely conceivable that a tabetic should be regarded as simply hysterical, for a single objective tabes symptom suffices to establish the diagnosis of tabes and exclude that of hysteria.

The danger is as slight of confounding tabes and hypochondria. From time to time young physicians and other persons, the sins of whose youth cause them anxiety, come for advice believing that they have incipient tabes. The absence of all objective signs generally renders the decision easy, although in such cases one cannot always make comforting assurances with respect to the future. It is unfortunately no rarity that a genuine tabetic is regarded as a hypochondriac. This happens only when the physician is ignorant or examines improperly. If nothing is found, but if the pains as described are those of tabes and if syphilis has preceded, the decision of the question must be simply postponed. To the expert the way in which the patients complain of their tabetic symptoms appears so different from the way of hysteria and hypochondriasis that generally he has no doubt from the first.

Under the unsuitable name "pseudotabes" a number of quite different conditions has been comprehended, and first, various forms of neuritis. Of these only the alcoholic neuritis may give rise occasionally to difficulty of diagnosis. If reflex iridoplegia is absent, no positive opinion may be possible for a time. Of course tabes and alcoholic neuritis may occur together. A recent tabetic, for example, with reflex iridoplegia and lightning pains may develop alcoholic

paræsthesiæ and paralyses. It is necessary then to proceed cautiously and to separate the etiological factors. The distinction between tabes and diabetes has already been mentioned. Really doubtful cases of this kind are very rare. The name "pseudotabes hysterica" is quite absurd. And the designation "pseudotabes syphilitica" is also to be rejected, for in the cases in which it has been employed the condition has been not a new disease, but rather tabes with meningitis syphilitica, that is, a complication in which analysis not synthesis is in place.

Diphtheritic paralysis may occasionally suggest tabes, especially if, exceptionally, adults are attacked and if the fact that diphtheria has preceded is not known. The patient may, for example, have weakness of the legs with paræsthesiæ and loss of the knee phenomenon, or in the severer forms paralysis of the eye muscles, anæsthesia, and ataxia may be present in addition. But reflex iridoplegia, the characteristic tabes pains, and the bladder disturbances are absent, paralysis of the palate has generally preceded, and finally the course of the disease makes the diagnosis clear.

In all cases in which the diagnosis is questionable, we must inquire whether the patient has had syphilis. In children and very young and very old persons tabes is possible, but extremely rare and therefore improbable. In women it is more improbable than in men. Conditions which do not favor the existence of syphilis (*e.g.*, virginity) likewise do not favor tabes. On the other hand in men of middle age, especially in those whose position and mode of life is such that a suspicion of former excesses seems justified, we should think of tabes from the first. Such patients should be examined for tabes, even if other diseases are present; and, on the other hand, the examination of the heart, the urine, etc., must never be neglected in tabes, since in connection with it atheromatosis, cardiac lesions, contracted kidney, and diabetes frequently occur.

Prognosis.

It is sufficiently well known that tabes is not curable and that its course varies greatly in its nature and its duration. The question of prognosis then presents itself as follows: Can we predict in a given case what course the disease will take? Unfortunately we cannot do this as a rule, yet there are some hints which may be of service. In general, the more slowly tabes begins the more benign it is. If moderately severe pains and slight disturbances of the bladder have existed for years, there is a certain probability that the tabes will continue to be benign. On the other hand, a more or less stormy begin-

ning is of evil significance. Early developing and rapidly increasing ataxia is especially unfavorable. Tabes dolorosa is a bad form of the disease in which severe and frequent pains not only bring the patient to despair, but also diminish his bodily strength and power of resistance. A similarly bad form, and indeed one of the worst, is the one with gastric crises. In this both pain and hunger assail the patient; it is necessary to feed him as well as possible in the intervals of the attacks, in order that he may not be worn out. Unfortunately when gastric crises are present, it is not rare for other crises, especially those of the larynx, to develop and make the prognosis still worse. Other symptoms are also of unfavorable omen from the outset, especially affections of the osseous system. Aside from the fact that a fracture or an arthropathy is in itself a serious matter and causes the patient much harm by confining him to bed, such accidents frequently occur in succession, because the disease of the bones is more or less general. Perhaps a good deal depends upon the patient's constitution and mode of life, at least it seems that severe accidents of this kind are more frequent in the poor than in those who are better nourished and able to spare themselves. A severe affection of the bladder is naturally of a bad prognosis. Sooner or later, with or without the aid of the catheter, infection of the bladder takes place, and there is the added danger of pyelitis. Not a few tabes patients succumb to pyelonephritis.

A symptom which in itself is among the worst, but in general promises a favorable course of the disease, is early developing atrophy of the optic nerve. Singularly enough the blind tabes patients as a rule become ataxic at a late stage or not at all, and suffer proportionately little in other respects. How this fact is to be explained is not known, but the fact itself is undoubted. But if optic-nerve atrophy appears at a late period when ataxia is already present, there is then no trace of its apparently inhibitory influence.

The simultaneous occurrence of gastric and laryngeal crises has already been mentioned. Similarly affections of the cerebral nerves may be combined. To the ophthalmoplegia may be added a lesion of the trigeminus, manifesting itself by pain alone, by pain and anæsthesia, or by the latter and loss of the teeth. The same remarks apply to lesions of the facial and auditory nerves.

Of course all paralytic symptoms, apoplectiform or epileptiform attacks, disturbances of speech, and psychical disturbances are of evil significance. It is known that progressive paralysis may appear even at a late stage of tabes. But often very early it sends forth its forerunners, such as quite transient attacks of aphasia, quivering of the facial muscles, attacks of ocular migraine, etc. Such

symptoms should be carefully considered, for they mean much. Not only does the paralysis usually end the patient's life at a comparatively early period, but even a mild form of it generally disables the patient from pursuing his business, while in simple tabes he can work undisturbed in many kinds of occupation.

Much of course depends also upon the complications. Probably the majority of tabes patients do not die of tabes but of other diseases—contracted kidney, apoplexy from atheroma of the vessels of the brain, valvular lesions or simple degeneration of the myocardium, etc. As the disease advances, the prognosis is more easily made. While at the beginning everything is possible, after some years the disease has generally in so far revealed its character that we can at least determine whether it is benign or malignant. Surprises are still a possibility, yet the rule is that a benign form continues to be benign.

The prognostic significance of the individual symptoms appears from that which has been said here and under the head of symptomatology.

In any case, the physician does well not to express himself too pessimistically. The patient has a right to encouragement, and the physician may point out the fact that the disease may improve or remain stationary, and should bear in mind that this may occur occasionally when the prospect appears exceedingly gloomy.

Treatment.

The history of the treatment of tabes is painful and touching. It shows us, on the one hand, how weak human judgment is, and, on the other, how much faith man possesses. New methods of treatment are constantly invented, and all are of benefit so long as they are new, but all in time lose their virtue and are then quietly cast aside. Yet it cannot be denied that any methods have attained positive results. Of course most of the curative effects, in so far as they are conscientiously reported, are in reality remissions which the natural course of the disease carries with it—that is, the periods of improvement which are observed in most tabetic cases, even without treatment, chance to coincide with a course of treatment. Still, cases are reported in all methods of treatment in which it is difficult to believe in chance, cases in which after a long, unchanging course of the disease a distinct improvement commences immediately after the institution of the new treatment. Since such curative effects are attained by all methods, the unreasonable as well as the reasonable, but one explanation remains, to wit, that here as elsewhere it is not the remedy but

the patient's belief in the remedy that benefits. Indeed, tabes is a conspicuous example of the fact that even affections which are apparently due to organic lesions may be influenced by faith. More correctly, we should say that experience derived from tabes shows that, even when organic lesions exist, a considerable part of the disturbances of function are of a psychical nature, for no one can intelligently assume that degenerated nerve fibres can regenerate themselves under the influence of faith. If the latter removes disturbances it must be that these disturbances do not depend directly upon the degeneration, but are accessory affections which the patient has suggested to himself, which anxiety, fear, and despair have added to the principal affection.

Progress has appeared in the course of time, inasmuch as the conviction has gradually gained ground that tabes is incurable. We may say that to-day all well-informed physicians are agreed that there is no cure for tabes, that all reports of cure are mistaken. Formerly it was of every-day occurrence that confident reports of cured tabes cases were published. One had cured tabes with galvanism, another with faradism, another by means of this spring, still another by baths in another spring. One had cured with mercury, another with iodine, another with silver, another with ergot, by nerve-stretching, or by homœopathy, and so on. Such bold assertions are still made indeed, but they are no longer believed by the well-informed. The therapeutists of to-day, however confident they may be, content themselves, as a rule, with speaking of improvement and are satisfied with even a transitory improvement.

I do not assert that no remedies have aoy physical effect whatever upon tabes. That cannot be proved, and it is possible that some of the drugs and methods now in use exercise a favorable effect upon the diseased parts, so that they resist the destructive influence for a longer time, or it is possible that some drugs strengthen the still uninjured fibres and cells so that the loss of function on the part of those already diseased is less apparent. But one should be very sceptical with respect to such conjectures; for that a remedy which has seemed to be of help in one case should help subsequent cases also, as might be expected, appears not to follow at all. Rather, on the contrary, the results are not at all to be predicted. Of two apparently identical cases, in the one the curative effect appears undeniable, while in the other no trace of improvement can be detected. Moreover, the individual symptoms seem to behave differently. The more objective a symptom is the less accessible does it appear to therapeutic measures. The degeneration is visible only at one place, in the optic nerve; but here only the most enthusiastic speak of the curative

effect of this or that remedy, every one else is convinced that all treatment is useless. Reflex iridoplegia always remains unchanged. Other symptoms, on the contrary, such as anæsthesia and especially ataxia, vary in severity not only during the natural course of the disease, but also during treatment. The ataxia is the most easily influenced, and that is easily understood, for it is only the reaction to the anæsthesia of the joints and of the deeper tissues in general. This reaction differs with the individual and with the varying condition of the individual. As we shall see subsequently, it can be changed by a kind of education—a proof that its degree depends upon the state of the unaffected cerebral cortex as much as upon that of the diseased spinal cord.

Consequently I regard it as probable that we cannot influence the tabetic process itself directly in any way. We may under certain circumstances influence the tabetic patient favorably, but this is effected partly because healthy portions of his economy are strengthened, partly because his faith helps him. Against tabes itself we are powerless. This is no quibble upon words, it is an important distinction. In the first place, it is unworthy of the physician to grope about with blind faith in his therapy, like the unthinking masses. When suggestion is at work he should be aware of the fact and take his course with the painful but emancipating knowledge of the weakness of his art. He will not then embrace with foolish enthusiasm every new remedy in order to abandon it as quickly. Above all things, he will spare the patient, because he knows what the future will bring. In such a tedious disease as tabes the physician cannot abandon his patient, he must treat him under all conditions and must employ various remedies and methods in order to keep up his courage. But, if he is properly skeptical, he will confine himself to what is necessary, he will abstain from officiousness and, last but not least, as an honorable man he will spare the patient's purse, if the patient is poor. How many people have spent their savings in useless and expensive treatment!

After these general remarks we will consider the modes of treating tabes which are now in use, passing by those which have become historical.

Is there a prophylaxis of tabes? Since tabes is metasyphilis, avoidance of syphilis is also avoidance of tabes. Here an important duty is set before the physician, which he has hitherto very insufficiently performed. Physicians should tell the people that syphilis is a fearfully serious matter, that an infection with it may ruin the whole life, and that there is only one means of avoiding it—the avoidance of all impure sexual contact. It is a crime to speak jestingly of

syphilis, to call it a "child's disease," to advise patients to have intercourse with prostitutes. Supervision of prostitution, the careful treatment of the syphilitic, and the severe punishment of those who carelessly or even knowingly propagate the disease are also important, but not much is to be accomplished by them. Many believe that by energetic and long-continued treatment of syphilis the development of tabes may be averted. That is an unproven and not very probable assumption. Whether by such treatment tertiary manifestations may be prevented, as many syphilologists believe, is another matter. Tabes is not a tertiary manifestation, and in fact it is even characteristic of tabes that it most frequently follows an attack of syphilis which has been without tertiary symptoms.

A. Fournier has said that the better the treatment of syphilis the less is tabes to be feared. But Fournier himself admits that tabes may occur after treatment of syphilis, which has been excellent, in his meaning of the word. He has himself seen such cases. He depends for the proof of his assertion upon the fact that in most cases of tabes the treatment of the antecedent syphilis has been insufficient, and he gives the following statistics: Of 312 tabes patients, there were 24 whose syphilis was not treated at all, 70 who had a three to six months' treatment with mercury, 74 who were treated for from six months to a year, 32 who were treated for from one to two years, and 13 who were treated for from two to four years. It is evident that such statistics can prove nothing. If one should question 312 persons who had had mild syphilis and were not tabetic, one would probably obtain quite similar figures. Manifestly most cases of syphilis are benign, and most syphilitics are treated insufficiently, in Fournier's sense. If such insufficient treatment were to blame for tabes, the latter would be far more frequent than it really is.

Although the results of the mercurial treatment are quite doubtful, it is still to be recommended to carry it out conscientiously and to combat every manifestation of syphilis energetically and persistently. Whether it is justifiable when no sign of syphilis is visible to prescribe mercurial treatment for from four to six years, the future must decide.

Prophylaxis directed against the accessory causes of tabes consists chiefly in the rules for health which apply to all. We may conjecture that one will be the more likely to escape tabes, notwithstanding the syphilitic infection, the more healthy he otherwise is, and the more he avoids the injurious influences which are in general prejudicial to health. Although it is very doubtful whether emotional excitement, mental and physical exertion, the use of liquor, and sexual overstim-

ulation help to provoke tabes when it is not yet present, yet every one who is syphilitic should be warned against these things even more than others. A special importance is perhaps to be ascribed to exposure to cold. This is met with so frequently in the anamnesis, the first pains have so often appeared after repeated exposures to cold or after a single but very severe exposure, that it is probably justifiable to especially warn candidates for tabes against such exposure.

Since syphilis is the cause of tabes, antisyphilitic treatment appears to be the causal indication. As a matter of fact, this was employed as soon as the connection between the two diseases appeared to be probable. The interest in the question as to the relations of tabes to syphilis probably depends for the majority upon the hope of now being able to attack tabes by means of mercury and iodide of potassium. But this is evidently to be determined solely by experience, for the question cannot be decided theoretically. Pathological anatomy shows that the anatomical changes in tabes are quite different from those of tertiary syphilis. But from the first it is clearly impossible that iodine and mercury could be curative in simple atrophy of nerve fibres. We know of course that the intraspinal nerve tissues which have perished are not regenerated. A cure by means of iodine and mercury must therefore appear excluded to him who is familiar with pathological anatomy. But it is quite conceivable that antisyphilitic treatment might arrest the tabetic process and effect a regeneration of the diseased peripheral parts, although even that appears doubtful when we reflect that in metasyphilitic diseases similar to tabes, which manifest themselves as sclerosis of the liver, kidney, and other glands, mercury and iodine are quite useless. Only experience can decide the question. And experience teaches that it is not possible to check tabes by means of antisyphilitic treatment. In this the great majority of authors are agreed, and even those who most highly commend antisyphilitic treatment in tabes venture to promise neither cure nor arrest of the disease, but only affirm that occasionally there has been "improvement." At the first, indeed, reports appeared from various quarters, announcing the complete cure of a tabetic patient, but for what new treatment have not enthusiasts made the same claim? Critical authors, however, have always rejected such assertions with unanimity and in part admit the entire uselessness of the antisyphilitic treatment, in part recognize that the success of that treatment is far from corresponding with their expectations. Erb has expressed himself most positively in favor of antisyphilitic treatment, but even he says "that our experience as yet with respect to the value of the antisyphilitic therapy in tabes is by no means brilliant, and indeed not very satisfactory."

Erb, through Dinkler, has published a large number of case histories which are intended to demonstrate the value of mercury and iodine. From this collection it should appear that "in six-sevenths of the cases a more or less distinct" and extensive improvement of the affection had been produced. But if one enters into the details of Dinkler's presentation of the subject it is seen that the results are quite similar to those which are attained by any treatment. Very similar histories are found in the electrotherapeutic literature in large numbers, not to mention the reports on suspension and nerve-stretching. As yet it has never been proved that antisyphilitic treatment can prevent the further development of tabes. Whether transitory improvement can be attained by it can hardly be proved, nor can it be disproved. In any event I regard it as probable that the antisyphilitic treatment is of no more benefit than the other modes of treatment hitherto in use, although in these also there is but little proof for or against them. In my own experience I have never seen any positive results from the treatment by inunction, and many other physicians have had the same experience. I cannot, however, deny the possibility of benefit; and this at least seems to me to be shown by the experience of Erb and of others, namely, that a judicious antisyphilitic treatment does no harm in tabes. It has been asserted several times that the atrophy of the optic nerve has rapidly advanced during the inunction treatment. But the same is seen when no mercury is used. The assumption that tabes is caused by the mercury is so ridiculous that it is not necessary to discuss it.

Well, then, inunctions do no harm and it is doubtful whether they do any good. In this state of things, the question is to be asked, When should inunctions be employed? Erb says "in the tabetic who have been syphilitic antisyphilitic therapy is indicated." I would prefer to alter this sentence to read, "antisyphilitic therapy is permissible in all tabes patients." If the view which I support is recognized that tabes is always metasyphilis, there are no tabetic patients who have not been syphilitic. In case of those who have apparently not had syphilis, inunctions are more distinctly indicated than in the others. Those who deny syphilis and those who for the most part actually know nothing of their infection belong quite certainly among the patients whose syphilis has been insufficiently treated, and it is all the more necessary to make up for lost time. In general, the treatment by inunction seems to me to be especially justified as an experiment where it has not been employed for a long time, or not at all. In such cases tertiary residues will also more probably be present with the tabes. That inunctions are indicated when tertiary symptoms can be demonstrated is a matter of course, but such cases

are very rare. Every physician will be obliged to use his own judgment in the individual cases.

I cannot recognize it as a duty to advise the inunction treatment under all circumstances, yet if it is desired to employ antisyphilitic treatment, inunctions are certainly to be especially recommended. Three or four grams of blue ointment are rubbed into the skin every day, a bath is taken on the seventh day, and the cycle is repeated four or five times. With this treatment care of the mouth is of course necessary. We must strongly warn against injections of compounds of mercury which are soluble with difficulty. In every case it is advisable to interrupt the treatment after six or eight weeks, and, even if a favorable result appears to have been attained, to give a second course of treatment only after an interval of several months. Erb recommends that the interval be occupied with a general tonic treatment by mountain air, mild hydrotherapy, especially sitz-baths at 24° to 18° R. (86° to 72.5° F.), the internal administration of tonics, electrical treatment, perhaps also the baths of Nauheim or Rehme. He thinks that this is important and that only after the termination of the mercurial treatment by means of these subsequent measures will the entire favorable effect be produced. In other words, the mercury really only lays the foundation for the greater and more certain effect of the other remedial measures. This appears to me a somewhat arbitrary construction, and I admit frankly that my faith is not strong enough to atone for my lack of insight into the relations of these things. I am also in doubt with regard to Erb's recommendation to supplement the mercurial treatment by baths at Aix la Chapelle or at Nauheim, or by electricity, etc., for, since according to Erb all these various remedial measures may be of efficacy, if the patient should really improve it would not be known what had actually benefited him. And finally, Erb's urgent admonition that the patient must give up business during the course of inunctions arouses my doubts likewise, for in the treatment of affections in which these really are of use the patients do not need to give up their work. On the other hand, rest is often very beneficial to tabes patients even without inunctions, so that the good results ascribed to the latter may really be due to rest.

In considering the large number of other "curative measures" for tabes, we may make a beginning with the drugs, other than mercury and iodine, which deserve notice, although but little that is good can be said of them. Since the time of Wunderlich silver has been used for tabes, why I do not know. The cases in which silver is said to have effected cures are quite old and few in number. For historical reasons, I have very often prescribed silver, but I have never been able

to convince myself that it had any curative effect. Nevertheless it does no harm, if argyrosis be avoided, and it is not impossible that it does some good. Since we cannot well spare a medicine for tabes which has some individuality and which can be given without harm for a few months, we always return to the silver salt. It is administered in pills (℞ Nitrate of silver, 1 gm.—gr. xv., white bole, 5 gm.—gr. lxxv. Ft. pil. No. 100. Sig. One pill three times a day), about two hundred pills being taken. It is dangerous to give more than this, because in many persons silver quickly causes argyrosis, and it is a serious mistake to produce this condition. After an interval of several months the course of treatment with silver may be repeated.

With the other drugs used in the treatment of tabes I have no experience. Ergot and arsenic have been especially recommended. It is known that in ergot-poisoning there may be an incurable disease of the nerves in which a part of the fibres of the posterior columns of the spinal cord is destroyed. It has been thought that if ergot can destroy the posterior columns, it can also cure them—an idea which appears ingenious rather than worthy of acceptance. Of late, moreover, those who extol the merits of ergot have been very silent, and it must be said that this poison which has been recommended in many nervous diseases on speculative grounds has been of benefit in none. Treatment with arsenic seems to be used in England. It will probably do no harm.

Strychnine, like ergot, is a laboratory remedy. Erb has again commended it of late as a remedy for tabes, but he adds cautiously that it may have simply a general tonic effect as a nervine.

No fault can be found with the administration of "tonics" in general in tabes. They are merely harmless, suggestive remedies. It would, however, require strong faith to believe that they have any beneficial effect upon the disease itself.

The modern humbug, organic extracts, is a repulsive subject to mention. We may well believe that physicians who have ventured to treat tabes with testicular juice or sheep's brains are now thoroughly ashamed of it.

Baths have always played an important rôle in the treatment of tabes. Formerly the indifferent thermal baths were chiefly used. Of late the majority of the German tabes patients who can afford it go to Nauheim or Rehme-Oenhausen, the French to La Malou. Experience seems to teach that hot baths are injurious to tabes patients, that consequently the springs the temperature of which is high are not to be recommended. But it is manifestly only the temperature that is to be avoided, for if the waters of Teplitz, Gastein, etc., are cooled to the proper temperature, about 27° R. (93° F.), they do no harm.

Besides the chief baths above mentioned, many others are visited by tabes patients with apparently good results—sulphur baths, like Aix la Chapelle, baths in the mountains, such as Pfäfers-Ragaz, Wildbad in Würtemberg, sea-baths, ferruginous baths, etc. It is of course very difficult to say anything as to the effect of the baths unless one indulges in pseudo-scientific phrases about the reflex influence of the skin, changes of the circulation, etc.

Treatment by baths is an old custom; we have at present nothing better to offer in its place. It is like the silver pills. We ask the question whether they do any good. We do not know, but if used properly, they do no harm, and since something must be done—*eh bien!* I have as yet never seen any curative effect after the use of any of the baths. Sometimes the patients have somewhat less pain afterwards, sometimes the ataxia is somewhat diminished—in short, it is like all other modes of treatment. Every one will admit that a stay at a bathing resort may have a stimulating and refreshing effect and that the baths may do the patient good. Every such bathing resort is a strong suggestive remedy. People see the water gushing from the earth, they convince themselves by sight, taste, or smell that it is no ordinary water. From antiquity the sick have made pilgrimages to the spring, and many traditions have sprung up. With their own eyes they see streams of invalids from all lands. One tells the other of wonderful cures, which he or his uncle has witnessed. Grave and dignified physicians confirm these stories and explain scientifically the importance of the spring. The state itself gives its sanction and the whole population of the locality swears to the value of the water upon which their livelihood depends. If many cases of the same disease are collected together at one place the physicians learn to know their needs accurately and everything is done that appears to be of benefit. So the physicians at Rehme and Nauheim, where during the season hundreds of tabes patients are to be found, have finally become specialists. The patients find good roads, sufficient resting-places, rolling-chairs, etc. Many patients return every year to this place. They return to their homes in general about as they went away, but yet they have done something for themselves. Hence I shall say nothing against the use of baths. On the contrary, I send patients there myself, because it is customary and because it does the patient no harm and possibly may benefit him. But here too there may be too much of a good thing. Many patients receive more harm from the fatigue and discomforts of the long journey than the problematical benefit of the treatment can compensate for. Not a few spend their hard-earned money, with which at home they could procure what would really

benefit their health, because they have been persuaded that they must by all means visit the baths.

The hydrotherapeutic institutions are formidable rivals of the baths. Even if one does not believe in the hocus-pocus which is designated scientific hydrotherapy, he must recognize the value of its empirically approved methods. As a mode of treatment of tabes, hydrotherapy is of course of as little value as the other methods, but as a mode of treating the tabetic patient it is of more importance than many others. These patients are very often confined to their rooms and are extremely sensitive to cold. A powerful stimulation of the nutrition of the skin and of the blood-vessels and a judicious hardening process are for them of great value. To be sure, that which is sought could be attained at home by rubbings with cold water, sitz-baths, douches, etc. But the institution affords at once the benefits of a summer resort and of a hospital managed by physicians. It is to be noted at the institute and at home that no very strong stimuli are suitable for tabes patients, that therefore not only high but low temperatures, powerful douchings, and the like are to be avoided. There is of course a difference between patients. What agrees with one is too strong for another, since among these patients, according to the original constitution and the development of the disease, there are strong and weak, and those who are sensitive as well as those who are excitable with difficulty. Especially in hydrotherapy it is necessary to individualize judiciously. For relatively strong tabes patients short sea-baths are very suitable. But for the majority is to be recommended only residence at the seaside without bathing.

Electrotherapy in tabes in my opinion is suggestive treatment. That this "mighty physical remedy" is of influence upon the tabetic process itself is so difficult to believe that a "*sacrificium intellectus*" is really necessary for faith. While in hydrotherapy and some other methods, at least a strengthening of the body as a whole may be effected, this advantage does not obtain in electrotherapy, for no one surely will assert that by the passage of a feeble current for a few minutes through the skin, muscles, and perhaps a few nerve fibres the body is strengthened. The electrotherapeutists claim that only when electricity is employed *secundum artem* can it be of benefit, and each holds that his own method is the correct one. I have occupied myself long enough with electrotherapy to have mastered the technique and I have employed the various methods of the authorities upon tabes patients, but I have never seen any effect that could not on good grounds be ascribed to suggestion. And many experts have had the same experience as myself. From year to year the psychical nature of electrotherapeutic cures becomes more widely recognized, and so

far as the electrical treatment of tabes is concerned many will agree with me who do not yet doubt the physically curative effect of electricity in general. I do not mean by this absolutely to condemn the electrical treatment of tabes, for media for suggestion are necessary and electricity is at least harmless if it is not used with actual brutality. I have seen great scars upon the backs of tabetic patients which had originated from unskilled galvanization and which with propriety might have led to suits for malpractice. A long electrical treatment of patients with small means may lead to an ill-afforded expenditure which does the physician little honor.

The following methods of electrization are used in tabes: One pole is placed upon the lumbar spine, the other upon the neck; one electrode is then slowly moved towards the other, and the somewhat childish idea is entertained that the current is thus made to pass longitudinally through the spinal cord. Others place one pole upon the "cervical sympathetic," *i.e.*, under the angle of the jaw, the other upon the back, or one pole upon the chest while the other is passed up and down the back. Some place the anode upon the sensitive locality (certain vertebræ), the cathode somewhere else and thus give a "polar" treatment. Broad plates are always used as electrodes. The sitting lasts a few minutes. The strength of the current is four, six, or eight milliampères. Some authors advise also galvanization of the limbs (anode upon the lumbar spine, cathode upon the nerve points in the legs, or anode upon the neck and the cathode upon the arms). I have done this also and the patients were generally most pleased by this application, because according to their view the limbs themselves are diseased.

The faradic current has found few friends, except that the faradic brush upon the skin of the trunk and of the limbs has been enthusiastically praised. The brush should be used for from five to twenty minutes and should cause distinct sensations but not actual pain. In this somewhat troublesome manner about the same effect is probably produced as by bathing with alcohol.

Such bathings or rubbings are quite useful as symptomatic remedies. They come under the category of cutaneous stimulants. But to see in these a treatment of tabes itself is an anachronism. The old physicians treated every possible internal disease with "derivation to the skin" from the crude idea that the materies morbi could be conducted to the exterior of the body. Hence tabes patients in former times were tortured frightfully. Their backs were burned with red-hot irons and with moxas, thus adding a new affliction to those already present. In modern times the implements of torture have become more refined. "Pointes de feu" are now used, and much mischief is

done with them, especially in France. The "pointes de feu" are directed to be produced upon a space the width of the hand along the spine about every ten days by means of a small thermocautery of from thirty to fifty points. The milder forms of cutaneous stimulation will be mentioned farther on.

Massage, which is unduly lauded by its advocates, is stimulation of the skin and something more. It is of course of no importance as a treatment of tabes, but general massage, like hydrotherapy, may occasionally be employed as a general tonic for tabes patients as well as for others.

Gymnastics, whether German or Swedish, are to be similarly regarded. But they have an especial value also as a remedy for the ataxia, to which we shall recur again. Treatment with Zander's machines is a mixture of massage and gymnastics. In some respects it accomplishes less than hand massage and individually conducted gymnastics, but it is not injurious if used in moderation and may occasionally be employed to advantage.

A chapter of modern medicine of which physicians should be ashamed is the doctrine of nerve-stretching and of extension of the spinal column. The idea of curing tabes by stretching the sciatic nerve is so absurd that every scientifically educated man would naturally repel it, but notwithstanding this it has not only been approved but has been enthusiastically praised, and the operation has been performed in a great number of cases. Even to-day there are physicians who are not ashamed to perform the operation or to allow it to be performed upon tabes patients. The operation consists in exposing the sciatic nerve in the upper part of the thigh, drawing it out of the wound with the finger or with a hook, and stretching it strongly. One or both nerves may be stretched and under some circumstances the crural nerves also. The fact that brilliant results were at first attained by the operation proves well how subject both the tabetic patient and his physician are to the power of suggestion. The reports of such successes are manifestly not all false, the patients really became better. But when the enthusiasm for the operation was dissipated by criticism and by reports of fatal cases the operation was no longer successful. The furor which had carried everybody away subsided and after a few years nothing more was heard of the matter. To one who has witnessed the rise and fall of such therapeutic enthusiasms, they appear like psychical epidemics and recall the dancing manias of the Middle Ages.

Just as the pointes de feu have succeeded the red-hot irons, so nerve-stretching without operation, and suspension have succeeded to the operative nerve-stretching. These are as absurd as the latter

but somewhat less dangerous. The non-operative nerve-stretching consists in flexing as far as possible at the hip joint the leg of the recumbent patient, the knee being extended. If anæsthesia and great relaxation of the muscles and ligaments are not present, as is sometimes the case in the tabetic, the procedure is very painful. If the patient sits on a firm table with outstretched legs and his trunk is bent forwards as far as possible the process is called "extension of the spinal cord"(!).

The procedure recommended by Bonuzzi is actually barbarous. It consists in drawing the feet of the recumbent patient by means of a cloth so far above his head that the knees of his extended legs approach the forehead. As extension *en miniature* we may regard the procedure of Blondell, who approximates the patient's bended knees to his chin and holds them in that position for a few minutes by means of a bandage passing behind the knees and about the neck.

Suspension, that is, hanging the patient in Sayre's apparatus, was first tried by Motschutkowsky of Odessa, then obtained the approval of Charcot, whose pupil Raymond had visited the Russian, and in consequence of this approval was received with general enthusiasm. At the Salpêtrière suspensions were done by wholesale. The patients came flocking in and the finest results were attained. The tabes patients of a large city, by the way, constitute a class always eagerly on the watch for something new. As soon as a new method of treatment is discovered, preferably one that is somewhat fantastic, they embrace it, and for a time the novelty is of benefit. Suspension was, of course, tested "scientifically." Some found that it produced a slight lengthening of the spinal column, others that there was on the contrary a slight shortening of it, rabbits and dogs were suspended, in short, the resources of science were employed in the investigation. Now, after a few years, nothing more is heard of it, at least in Germany. From time to time a tabetic says, shrugging his shoulders, "You know the time when we used to be suspended." The fact may have contributed to chill the enthusiasm that sometimes the heart of the suspended patient stops beating. In the original method the patients were suspended by the chin and neck and also by bands passing through the axillæ, and allowed to hang for several minutes, a procedure which might prove dangerous, especially in heavy patients and in those who had arteriosclerosis. Subsequently an apparatus invented by Dr. Sprimon was recommended, by means of which the patient, who is in a sitting posture, is seized by the chin and neck and by the elbows and raised by a weight (about one hundred pounds) suspended by a pulley. Some place the patient upon an

inclined plane with head and shoulders fixed and leave "extension" to the bodily weight, from which no harm can result.

The corset of the orthopedist Hessing, like suspension, is to be regarded as a suggestive treatment. This is supposed to support the vertebral column, and to relieve it of superincumbent weight; it is properly applied only after many trials, and costs enormously. Some tabetics really feel much more comfortable if they wear a corset (perhaps on account of its uniform warmth), but they can have one applied by any one skilled in bandaging.

The methods hitherto discussed hardly deserve to be taken seriously, but not so with the treatment of the individual symptoms, although by such treatment we do not expect to influence the tabetic process itself. Most frequently the physician is obliged to treat the pains of the tabetic. Formerly we were almost entirely restricted to external applications, but of late we have made an advance by the knowledge of new and rather harmless remedies for pain. The most important of these are acetanilid (or antifebrin), phenacetin, and antipyrin.

Besides these may be used sodium salicylate, salipyrin, exalgin, lactophenin, and a series of similar products which the activity of the pharmacologists has furnished us; ½ to 1 gm. (gr. viii.—xv.) of antifebrin may be given, the same amount of phenacetin, 1 to 2 gm. (gr. xv.—xxx.) of antipyrin, and 1 to 3 gm. (gr. xv.—xlv.) of salipyrin. Combinations of these can also be employed. All these medicines relieve the so-called "nervous" pains in an unknown way, probably by their action upon the parts of the brain which are connected with sensations of pain. It is folly to prescribe the bromides for the pains; they are of no use whatever. It is generally possible to relieve the lancinating pains by means of antifebrin and kindred remedies. Nevertheless we cannot dispense with external applications. For medicine should not be given for various reasons if it is not absolutely necessary, and, on the other hand, unless injurious doses are given, pains are often not so completely relieved by internal medicines that auxiliary remedies can be dispensed with.

Sometimes no treatment is necessary. All tabes patients do not have severe pain, and a certain proportion of them learn to endure their pain without assistance. If that cannot be done, a cold compress is the most simple and often an efficient remedy—much more rarely warm or hot compresses. Wet-packs, the so-called Priessnitz compresses, occupy an intermediate position. Then follow the stimulating applications, such as spirit of soap, of camphor, or of mustard, petroleum ether, oil with chloroform, or chloroform alone. My pa-

tients like paprika tincture, which I give in combination with spirit of camphor or of mustard. Remedies which easily injure the skin appear to me less advisable; such are mustard plasters, sprinkling chloroform upon hot compresses, and painting with tincture of iodine or solutions of nitrate of silver. Wounds of the skin should be carefully avoided. This objection applies also to sprays of methyl chloride, which for a time were highly commended. If they are used cautiously so as to produce no eschar, they are not, however, objectionable. Sometimes strong pressure helps, laying on sand bags, or bandaging on a lead plate, or constriction with cords. If everything fails and the pain appears to be unendurable, nothing remains but to use morphine, which always relieves. Since, however, in a disease of such long duration as tabes the danger of the morphine habit is particularly great, this drug should always be avoided, if possible. If injections are needed, they should be given by the physician himself. Singularly enough, patients rarely hit upon the idea of buying a syringe and morphine solution for themselves. It is only when the physician entrusts the syringe to them or to their friends that they become victims of the morphine habit. Yet here also we must individualize. Whether one who takes morphine contracts the habit or not depends upon his character. I know some tabes patients who are compelled to take morphine occasionally for their pains, who have not acquired a fondness for it and do not exceed relatively small doses. Although the use of morphine for the lancinating pains should be avoided as much as possible, the case is different with the so-called visceral crises, and especially with the gastric crises. Here only injections of morphine are of benefit; to give anything else is a loss of time; and in such cases as much morphine should be given at once as is necessary in order to subdue the pains, for divided doses are of no use. The other crises are much more rare and do not often give occasion for interference. The laryngeal crises are generally over with before the physician arrives. If necessary the application of cocaine by means of a brush may be tried.

In treatment of the vesical disturbances the most important thing is not to do harm, *i.e.*, do not catheterize except in the most extreme necessity. Many tabes patients have acquired cystitis and pyelitis through careless catheterization. In the great majority of cases the catheter is quite superfluous, for it is usually only necessary to wait, even in complete retention of urine. In case of relaxed abdominal walls, and in the later stages of the disease the bladder can be emptied by manual pressure. In bladder affections of moderate severity quite good results are often obtained with tincture of nux vomica in doses

of ten to twenty drops three times a day. Of course other preparations of strychnine may also be used.

As a rule, the sexual disturbances demand no treatment. In the rather rare cases of excessive sexual excitement bromide of potassium and cold compresses may be employed. Generally there is sexual weakness or impotence. In this case nothing is to be done, for it is quite unnecessary that tabes patients should have coitus or beget children, and even should the latter be desired for special reasons, we have not the power to be of assistance. Many physicians wish to oblige patients who have a desire for sexual pleasures, and with that object in view many baths and much electricity and medicine are given; this is also not one of the least reasons for employing the suspension treatment. But none of these things is usually of any avail.

Any mild aperient may be taken for the constipation which is commonly present. This may be continued for many years without harm if the remedy is changed from time to time. Electricity and massage, etc., are expensive and do no good.

Decubitus, perforating ulcer, and other affections of the skin, fractures, and arthropathies are of course to be treated according to the rules of surgery—of conservative surgery.

It is rarely necessary to treat the paræsthesiæ. If the patients demand treatment external applications may be tried. All sorts of cutaneous stimulation may also be employed for the anæsthesia, but generally there is no occasion for interference. On the other hand, the patients generally demand help for the affections which are due to the anæsthesia, especially the ataxia. The best remedial measure for this is systematic exercise. Many a thoughtful physician has no doubt advised exercise for the ataxic tabes patients, and I have known patients who of themselves learned how to suppress their ataxia by exercises, but it is only recently that the subject has been methodically approached. Frenkel first recommended exercise as a formal treatment of the ataxic. Quite material benefit, in fact, may be attained by it, which is not only good for the patients but very interesting theoretically. In all the methods of treating tabes their advocates have always called attention especially to the fact that by means of them those who were completely ataxic had learned again to walk. They shake their heads when assured that this is the effect of suggestion, for how could suggestion act upon the posterior columns upon the degeneration of which the ataxia depends? The answer is that neither suggestion in general nor Frenkel's method acts upon the posterior columns or upon the "coördinating paths," but upon the cerebral cortex. Upon its activity depends the accuracy of the voluntary movements, and exercise of it restores, so far as that is possible,

the accuracy of movement when disturbed by insensitiveness of the joints, etc.

The French have very appropriately termed Frenkel's method re-education; in fact, it is a mode of education. Its essence is that the patient learns to execute slowly and correctly with the aid of the eyes certain prescribed movements. At first, the patient being recumbent, he is required to raise each leg in succession to a certain height, to flex, extend, abduct, and adduct his legs, to place one foot over the other, describing larger or smaller areas, to touch the knee with the heel of the other foot, to describe circles and squares with the foot, etc. Then follow exercises in standing with legs apart and with legs close together, with one foot in advance of or behind the body, in sitting down and in rising, in bending the knees and in straightening them, in standing upon one foot and upon tiptoe. Finally come exercises in walking in time with measured steps, if necessary with the support of "running poles" (a kind of parallel bars) or of the arms of others, or with one or two canes, in "walking a line," and in walking forwards and backwards. The exercises should not exhaust the patient, and ought therefore not to be continued too long, especially at first, but may be repeated several times during the day. It is much more rarely necessary to prescribe arm exercises than leg exercises, ataxia of the arms being rather rare. But in this case the multiplicity of exercises possible is much greater and more scope is given for the ingenuity of the physician. Frenkel gives a large number of exercises: simple movements of the hands and of the fingers, drawing lines, describing figures, catching swinging balls, putting pegs into holes, counting money, writing, etc. It is desirable that all the exercises should be performed under the supervision of the physician or at least of some intelligent person who understands the object in view. The result depends upon three conditions, to wit, the skill of the teacher, the character of the patient, and the kind of tabes.

Very naturally strength of character and mental ability in the patient are of importance; a clever and persevering patient will gain more than one who is stupid and indolent. And, on the other hand, from the outset a strong-willed and self-reliant man will not give way to the ataxia so much as a weak-minded or nervous individual, who is open to unfavorable suggestions. Frenkel's treatment will manifestly attain the most brilliant success in the case of those who have taken up arms against the ataxia in good season. It becomes evident during the treatment of those who give themselves up to the disease, so to speak, in fear and despair, how large a part of their motor disturbances are of a psychical nature.

Amblyopia or blindness is an obstacle in the way of improvement. Fortunately blind tabes patients are not often ataxic. A distinction should be made between acute progressive ataxia and the common form which develops slowly. The former, as is known, disappears in part spontaneously after a time. So long as it advances there is not much to be done; rest is the best thing for it. If treatment happens to begin as the natural improvement sets in, it is apparently brilliantly successful. Only chronic ataxia affords really a good test of the treatment.

Before the tabetic paralyses the physician is almost powerless. As has been already explained, we have to distinguish between the transitory and the permanent paralyses. The former, especially the transitory paralyses of the ocular muscles, are apparently tractable under treatment, that is, if the patient, or even the physician, does not know their natural course, mercury or iodide of potassium, or strychnine, or electricity may appear triumphantly successful. That this success is deceptive is seen from the complete inefficacy of the same remedies in the permanent paralyses.

Treatment is also quite powerless in atrophy of the optic nerve. It may advance rapidly or slowly, or it may become stationary, but there is no true improvement. Long-continued injections, beneath the skin of the temporal region, of the cyanide of gold and sodium or of the cyanide of mercury have their advocates still, but I fear that they suit the physician better than the patient.

If the physician who is consulted by the tabes patients recognizes the disease, he should not mention its name to the patient. Generally the patients come with the idea that disease of the spinal cord condemns them with certainty to an agonizing death. I am accustomed to say to them: "You have a chronic nervous disease, which cannot be perfectly cured, because in it some nerve fibres are destroyed. But if you live prudently you may in all probability keep at work even if you are ill, and you may live long." Rules for the conduct of the patient's life are then to be given. Exposure to cold and overexertion must be avoided. The patient must have a dry and if possible a sunny dwelling. If his occupation exposes him to cold or to the risk of excessive bodily exertion, he should seek other employment. Warm clothing is necessary in cold weather. The feet especially should be kept warm. Fatiguing hunting, mountain-climbing, and long-continued riding must be given up. In all his activity the rule should be not to hurry and never to go further than the beginning of fatigue. Alcoholic beverages are to be taken sparingly. Abstinence is best, but is not always to be attained, and, according to our present knowledge, small amounts of alcohol do no more harm

to the tabetic than to the healthy. Tea and coffee are to be allowed. A few mild cigars are also permissible, for it is wrong to forbid more than is absolutely necessary. The patient may eat what he likes and can digest. If the patient is married, he may indulge in coitus if nature prompts. Emotional excitement is injurious to every one, especially to every invalid, and for the tabetic to refrain from worry and excitement is often of more value to him than medicine. Life in the open air is very beneficial. The rich may spend the winter in a mild climate, which enables them to live more out of doors.

Other psychical treatment is partly direct and partly indirect. Hypnotic suggestion is seldom indicated in tabes patients, because by it only individual symptoms, as the pains and ataxia, are influenced and because the majority of the patients, educated men of more or less advanced age, are but little receptive of suggestions. If susceptible individuals are met with, a few observations show whether good results may be obtained. Yet for the ataxia I would advise in preference the education already described. The pains come so irregularly that they cannot be anticipated, and if already present, they generally interfere with direct suggestions.

A certain general education of the patient is important. The physician should try to make it clear to him that it is better to endure the inevitable wisely and patiently than to lament and constantly to seek new remedies. If he is tolerably comfortable he should not be taking medicine constantly, and for that reason he should know that a complete cure is impossible. This the physician should tell him frankly, but of course to encourage hopes of improvement is not only permissible but necessary.

Aside from the symptomatic treatment, medicinal treatment serves as a means of indirect suggestion. In this the physician should give *non nocere* the first place. A simple hydrotherapy, the treatment customary at bathing resorts, and the administration of harmless medicines are chiefly to be recommended. I know some intelligent tabes patients who visit a spa or the seacoast or some other summer resort every year, and who take medicine to combat the pains, but abstain from all other treatment and keep relatively comfortable.

THE COMBINED SYSTEM DISEASES OF THE SPINAL CORD.

BY

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ERLANGEN.

THE COMBINED SYSTEM DISEASES OF THE SPINAL CORD.

EXPERIENCE teaches that the diseases of the nervous system may be divided with respect to their localization into two great groups—into systematic and non-systematic (diffuse) diseases. By systematic diseases (or system diseases) we understand those affections in which only nerve cells and nerve fibres, that is, neurons, which have a definite physiological function are attacked. The aggregate of all the neurons which subserve a definite physiological function is called a neuron system. And in classifying them according to our point of view we may distinguish general and comprehensive neuron systems and subdivisions of these, or special neuron systems. Thus, for example, we may group all the sensory nerves, that is, all those neurons which conduct external stimuli from the surface of the body to the seat of our consciousness, collectively in one great sensory neuron system. But in this great sensory system we may distinguish numerous subdivisions (the optic neuron system, the auditory neuron system, the neuron system for sensations from the surface of the skin, etc.), and each of these subdivisions may be still further divided (neuron systems for the sense of touch, for the sensations of temperature, for the individual primary colors, etc.).

The interesting fact that sometimes only the neurons which belong to a certain physiological system become diseased, all the other neurons remaining perfectly normal, is to be explained, so far as our present knowledge goes, as follows:

First, the clinical facts show that certain neuron systems are abnormally weak and sickly in some persons from birth. What the primary cause of this abnormality is we do not know. But very frequently it presents itself most distinctly as a hereditary or family affection. The highest degree of such a congenital abnormality manifests itself in the total failure of development of a certain neuron system. Thus, for example, cases occur of congenital spastic paralysis of the extremities which are due to the fact that the pyramidal tracts (the central motor neurons) have not developed at all (the so-called agenesis of a neuron system). But in other cases the neuron

systems in question are at first apparently normally developed and functionate in a perfectly normal way. Yet sooner or later in the course of the patient's life they are affected by a slow degeneration (atrophy) without the intervention of any special external cause. We may suppose that such neuron systems, abnormally weak *ab origine*, are injured and worn out by the very exercise of their functions, or that the mechanisms within the organism which should supply the constituents of the organ which have been consumed in its functionating are insufficient.

The second group of causes upon which the development of neuron system diseases depends are external in their nature. We know that certain chemical substances act upon the organism as poisons, that is, that they have an injurious influence because of their chemical action upon certain constituents of the organism. In consequence of the peculiar chemical nature of the poison such injury is often inflicted only upon certain definite cells of the organism, and these, as experience shows, are often well-defined neurons, that is, the neurons of a certain physiological system. Thus we know that lead has a toxic effect only upon certain motor neurons, that morphine acts only upon certain sensory neurons, etc. We know with regard to many poisons that, if long continued, they may so damage certain neuron systems that the latter may degenerate completely. The poison of ergot injures certain sensory neuron systems and hence produces systematic degenerations in the posterior columns of the spinal cord. The poison of spoiled Indian corn (in pellagra) and that of lathyrus (one of the Leguminosæ) if taken for a long time often cause degeneration of motor neurons (degeneration of the lateral columns of the spinal cord).

But most common of all is the action of toxins which originate within the body, almost always in connection with infectious diseases. Such toxins, the presence of which has as yet been positively proved in but few cases, similarly to the above-mentioned poisons, act directly upon the neurons, attacking chiefly certain definite neuron systems. The toxins of numerous acute infectious diseases affect particularly the peripheral motor neurons (so-called neuritis after typhoid fever, etc.); other toxins, such as the toxins of diphtheria, attack sometimes motor neurons (diphtheritic paralysis), sometimes centripetal sensory neurons (diphtheritic ataxia), causing degeneration. Of the greatest practical importance are especially the postsyphilitic toxic system diseases, of which degeneration of the peripheral sensory neurons (the so-called tabes dorsalis) is very common, while postsyphilitic toxic degeneration of motor neurons (postsyphilitic sclerosis of the lateral columns) is of far rarer occurrence.

We see, accordingly, that we must divide all system diseases into two great etiologically different groups: first, the endogenous system diseases due to abnormal congenital development; and second, the exogenous system diseases caused by external injurious influences chiefly of a toxic nature.

The endogenous system diseases all present the following peculiarities: first, they appear very often as hereditary or family diseases, which of course does not mean that the abnormal development may not occasionally manifest itself in an isolated individual. Secondly, they generally make their appearance in early life, often in children, or at least within the first two decades. The more careful the investigation the more early is the beginning of the affection found to have been. In isolated cases, however, the abnormal development may first become manifest only in advanced life. Thirdly, they generally have a very slow but steadily progressive course—at least within the region which is attacked—and are incurable, that is, they cannot be sensibly influenced by any external means.

The exogenous system diseases, according to the nature of the toxic influence, may develop in widely different ways. They may sometimes be acute in their development (as, for example, certain paralyses and ataxias after acute infectious diseases) and are then not rarely completely curable, the neurons being only in part damaged and recovering again their normal constitution and extent. Or they pursue a chronic course and are incurable because the neurons are completely destroyed and cannot of course be re-created by any means.

We may assume that it is completely proved for all system diseases that the affection always has its origin in the neuron itself and not in the glia or connective tissue, or in the blood-vessels and the like. The changes which are found in all systematic diseases in the interstitial tissues and in the blood-vessels are certainly of a secondary nature, having arisen in consequence of the primary destruction of the nerve tissue itself. How the disease develops in the neurons has not yet been accurately determined, but the kind of disease is always the same; it consists in a slowly or rapidly advancing atrophy (degenerative atrophy) of the nerve elements themselves, which has no inflammatory character. Numerous facts go to show that the visible degeneration generally begins in the terminal ramifications of the neuron (in the axis cylinder and the nerve processes) and only gradually approaches the centre of the neuron, the nerve cell in the narrower sense of the word. In some cases the nerve cell itself may also perish, in others it may be preserved for a long time. Occasionally the process even appears to attack the nerve cell at a relatively early

period, before its peripheral processes have degenerated. The latter then become diseased secondarily.

On account of the fact just mentioned that in all system diseases the neuron itself is the point of origin of the degeneration, it has been recently proposed to give up the expression, "system disease" and to substitute for it the term "neuron disease." I cannot regard the proposed change a happy one, for since the nervous system in general, aside from the interstitial tissue, is composed solely of neurons, every disease of the nervous organ is as a matter of course a "neuron disease." We might then speak of "primary systematic neuron diseases" in contrast with the diffuse and secondary neuron diseases, but instead of this we use, what is in reality a more accurate and suitable name, the briefer designation "system diseases."

The system diseases of the nerve organs are divided into simple and combined system diseases, which means that in some forms of such disease only one neuron system, or at least neuron systems of the same physiological function, are attacked; in others several neuron systems of different physiological functions. It is necessary to add this last qualification, because a system which is physiologically a unit may consist of several adjacent neurons. If several of such neuron systems which belong together, physiologically speaking, become diseased, we call the disease generally a simple not a combined system disease. Thus, for example, the corticomuscular paths consist, as is known, of two groups of neurons, of which the first, the central group, extends from the motor region of the cerebral cortex to the spinal cord, and the second from the motor ganglion cells of the spinal cord to the muscles. If these two related neuron groups become diseased, as is the case in amyotrophic lateral sclerosis, we call it a simple system disease (of the great corticomuscular conduction system). It is different with diseases which, for example, affect simultaneously systems of the posterior and of the anterior root zones (combined diseases of the posterior and lateral columns and the like). Here there is a simultaneous implication of neuron systems of quite different physiological significance and hence such diseases are rightly called combined system diseases.

Some system diseases occupy, as it were, an intermediate position, tabes dorsalis especially. This depends essentially upon a degeneration of the centripetal peripheral neuron system which enters the spinal cord through the posterior roots. In this sense it can be regarded as a simple system disease. But the peripheral neuron system of the posterior roots is much more complex in its physiological relations than that of the anterior roots. In the posterior roots and their continuations run fibres which subserve the functions of sensibility,

coördination of movements, and innervation of the bladder and rectum. And further neuron systems are also very often diseased in tabes which innervate the pupil or belong to the optical system (nervus opticus), etc. Tabes is therefore really a very complexly combined system disease, and it is only from the point of view of the centripetal conduction of all the affected systems that we are able to consider tabes as a simple system disease or a part of the great centripetal conduction system which proceeds from the periphery of the body. Because of this conception of the disease, tabes will not be treated of in this article, but separately.

If we omit tabes, there remain the following forms of combined system diseases which are to be more fully discussed in the following pages.

I. Endogenous combined system diseases: 1. Hereditary or familial ataxia, the so-called Friedreich's disease; 2. Hereditary or familial spastic spinal paralysis.

II. The exogenous combined system diseases of the posterior and lateral columns of the spinal cord.

Hereditary Ataxia (Friedreich's Disease).

Hereditary ataxia occurs with special frequency in brothers and sisters, whose parents or other ascendants have not had the disease. It is comparatively rarely that the disease is found in members of the same family in different generations, in which event it is frequently not quite typically developed. Whether any especial external moments are of influence upon the origin of that peculiar congenital predisposition which subsequently manifests itself as an early slow decay of certain neuron systems, we do not know. In most cases the parents of children who have Friedreich's ataxia appear to have been perfectly normal and healthy. But it has been thought that especial weight might be given to certain conditions, such as blood relationship of the parents, unusual difference in the ages of the two parents, syphilis or alcoholism in one of them, a general predisposition to nervous diseases, and the like. But a great significance should not be ascribed to any of these conditions.

The reason that the disease occurs far more frequently in the brothers and sisters of a family than by heredity in different generations may be in part that patients with Friedreich's disease rarely marry. But I know a patient with very severe Friedreich's disease who is married and whose children as yet are all healthy.

Nor should we by any means think that the disease occurs only in family groups. Isolated cases which are quite characteristic are not

rarely met with in persons all of whose brothers and sisters continue to be perfectly healthy. Such isolated cases have been observed with especial frequency of late, now that a knowledge of the disease and the ability to diagnosticate it correctly have become more widely disseminated.

From the earlier cases it seemed that the female sex was more predisposed to the disease than the male sex; but this appears to have been purely a matter of chance. If a large number of cases are examined, it is found that males are at least as frequently affected as females—from my own experience I might say even somewhat more frequently.

It is not easy to answer the question at what age the symptoms of the disease, as a rule, first appear. The disease develops with such extreme slowness that the patients, especially as they are often children, hardly notice its first beginnings. The time at which the more marked disturbances of the common acts of walking, eating, dressing, etc., first appear is consequently generally fixed as the time of the commencement of the affection. But the more careful the investigation the earlier is the first appearance of the disease found to have been. Sometimes the parents state that they have never had a really normal past, that they have always fallen easily, have always been clumsy in their movements, and the like. In such cases, then, the beginning of the disease dates back to the earliest youth. On the other hand, cases also occur in which the first manifestations of the disease appeared in the sixth to the tenth year, and it is even frequently the case that the beginning of externally conspicuous symptoms is assigned to the years of puberty (the thirteenth to the sixteenth year). Cases have even been reported which developed in far more advanced years (the fortieth to the sixtieth year). But such cases cannot be regarded as belonging to the typical form of Friedreich's disease.

Etiology.

Nothing, as a rule, can be found, if a search is made in the individual cases for external etiological factors. The disease has developed very gradually "of itself." But not very rarely an external exciting cause appears to be of some importance, above all the occurrence of some acute disease. With surprising frequency the parents state that they first noticed the abnormal gait immediately after scarlet fever, typhoid fever, influenza, measles, or the like. Yet if we inquire more closely, we sometimes learn that the disease was probably present in a very mild form previously, and that the new acute disease has merely led to its more rapid development.

Symptoms.

The cardinal symptom of Friedreich's disease, from which the disease receives its name, is the ataxia—a common ataxia, a disturbance of the coördination of movements, just such as occurs in tabes dorsalis, in many cases of multiple sclerosis, and in numerous other diseases. The extent and degree of this ataxia are its characteristic features.

The ataxia begins, as a rule, in the lower extremities and in the trunk; whether earlier in one than in the other of these localities, I am unable to say. It is almost always first recognized by changes in the ability to stand and to walk. The patients lose the power to stand steadily, and acquire an uncertain, tottering gait. If a patient with Friedreich's disease is required to stand erect and motionless with his feet close together, it is found that he does not stand quietly but that his body oscillates more or less (plastic ataxia). If the disease is further advanced, the patients can no longer stand at all with feet together but only when their feet are wide apart. With further progress of the disease standing at all is impossible. In order that the body as it sways may not fall, the patients, so long as they are able to stand alone, are obliged to make continually correcting muscular contractions. If, therefore, an unclothed patient with Friedreich's ataxia is observed while standing, continuous fine contractions with prominence of the tendons are noticed in the muscles of the legs, especially in the muscles which move the feet.

When the patient's eyes are shut, in my own experience the swaying of the body is as a rule increased (the so-called Romberg's symptom). Other observers have failed to find this phenomenon in Friedreich's ataxia. I do not believe that there are any fundamental differences here. The majority of patients who suffer with ataxia of the legs or of the trunk for any reason become accustomed to give their movements an increased degree of certainty by means of constant control with the eyes. Hence it is noticed that most patients with Friedreich's ataxia as a rule hold their heads bent forward in order to follow their movements with the eyes. If their eyes are shut, this control is lost and at the same time there is a subjective feeling of anxiety and of greater insecurity, and the oscillations of the body are increased until there is danger of the patient's falling. There has been too much theorizing in my opinion about Romberg's symptom in general. This depends, as observations in Friedreich's ataxia teach, neither upon anæsthesia of the soles nor upon a disturbance of the muscular sense, but is simply the consequence of the loss of the

control from the sight, which somewhat diminishes the manifestations of all ataxias. The degree in which Romberg's symptom is manifested depends upon the degree of the ataxia and upon the individual habit of the patient of utilizing his sight for the improvement of the ataxia.

The ataxia declares itself as distinctly or even more distinctly in walking than in standing. The patient gradually becomes more and more tottering, unsteady, reeling in his gait, until finally he cannot walk at all without the assistance of others. Even the first observers of Friedreich's disease noticed that the gait of hereditary ataxics does not precisely correspond with the gait of most tabetics, but rather with that of patients with cerebellar affections ("cerebellar ataxia"). The difference consists in this, that the tabetic has generally the power of walking in a straight line, but the movements of his legs in walking resemble flings and kicks. The movements of the legs are ataxic while the trunk is carried firmly and securely upon the thighs. In Friedreich's ataxia—and this is in fact very characteristic of it—the ataxia of the legs is from the first combined with ataxia of the trunk. Hence the patient's whole body reels and sways as he walks. His gait resembles that of a severely intoxicated person.

At the same time the movements of the legs themselves are ataxic, as can be seen, if the patient is examined in bed and is required to perform the knee-heel test (touching one knee with the heel of the other foot) or other complicated movements with the legs. But the fact is above all characteristic for Friedreich's ataxia that the ataxia very soon makes its appearance in the upper extremities. Hence all the more complex movements of the hands (sewing, writing, dressing, etc.) become more and more difficult and finally impossible. If the patient is asked, his arms being extended, to bring his index fingers together, or to touch with the finger objects which are held before him, the ataxia becomes most distinctly apparent.

In general, the ataxia first shows itself in the trunk and in the lower extremities and only subsequently involves the arms. But cases occur in which the unsteadiness becomes apparent in the arms at an earlier time than in the legs.

Besides the ataxia two other symptoms are to be mentioned which Friedreich himself regarded as other special localizations of the ataxia, viz., disturbances of speech and nystagmus. The disturbances of speech are supposed to depend upon an ataxia of the speech muscles, and the nystagmus upon an ataxia of the eye muscles. Whether this view is correct, further investigation will decide, if it acquaints us with the as yet entirely unknown anatomical bases of these two symptoms.

The speech disturbances appear almost always only in the more

advanced stages of the disease. Sometimes they may be entirely absent or very slightly apparent. The speech becomes slower, sometimes scanning, the articulation indistinct and confused, the modulation of the voice monotonous. Some syllables are uttered rapidly, others are much prolonged.

The nystagmus also first appears after long duration of the disease. It manifests itself not only during the movements of the eye, but also during the (especially lateral) fixation of an object. According to my own experience nystagmus is rare and certainly not characteristic of the disease. In cases in which it is not detected, it is said that it may be produced, if the patients are caused to revolve passively a few times about the axis of their own bodies. This phenomenon has not been observed in my patients.

As a rule, the power of the muscles is completely preserved for many years. Even with the most marked ataxia, as in the tabetic, there is often no evidence of true muscular paresis. But if the disease continues to advance a decided weakness (paresis) of the muscles, especially of the legs, does finally develop. The movements become feeble and are more readily checked by passive resistance. Hence the gait of the patient now assumes a somewhat different character; it becomes ataxic-paretic, the knees give way easily and the legs are dragged in walking. Walking now often becomes impossible. Whether a true paralysis (paraplegia) ever results in Friedreich's disease, I do not know. As a rule, the process does not advance beyond a simple paresis.

Paralyses within the region of the cerebral nerves (eye muscles, etc.) have as yet not been observed in hereditary ataxia.

The nutrition and the volume of the musculature remain completely normal for a long time, as well as the electrical excitability. It is worthy of note that many patients have always had a weak, poorly developed musculature. I have been especially struck by the deficient development of the leg muscles. As we shall see farther on, pronounced muscular atrophy may be combined with hereditary ataxia. In one of my cases the great, certainly congenital, thinness of the toes was noticeable.

The tonus of the muscles, in general, is not so diminished as in tabes. Passive movements not infrequently encounter a certain muscular resistance, but contractures do not generally occur. But a marked position of hyperextension in the great toe and occasionally also in the other toes is often noticed. The development of clubfoot —by no means a common complication—is probably always connected with paresis and atrophy of the peroneal muscles. In this connection it may also be remarked that the spinal column shows remarkably

often a slight scoliotic or kyphoscoliotic curvature. I am of the opinion that this is, to a certain extent, a sign of degeneration, a stigma of defective development, as is the case with the not infrequent scoliosis of patients with syringomyelia. As Friedreich himself insisted, the sensibility of the patients is entirely, or almost entirely, unaffected. This fact of the development of an uncommonly marked ataxia without a simultaneous diminution of the sensibility of the skin and of the muscles is of great theoretical importance, because it proves with certainty that ataxia is not the result of a disturbance of sensibility. Notwithstanding the most careful examination, I have been unable to find any abnormality in the cutaneous and muscular sensibility in several patients with very severe hereditary ataxia. But that does not mean that disturbances of sensibility never occur. On the contrary, in advanced cases careful examination often discloses slight disturbances of the cutaneous sensibility, especially in the ends of the extremities (hands and feet), sometimes also slight disturbances of the muscular sense. More pronounced anæsthesia, as in tabes, is certainly quite exceptionally if ever present. Pains and paræsthesiæ are likewise generally completely absent, having been observed in isolated cases only.

The examination of the reflexes is of great importance. While the cutaneous reflexes of the soles of the feet are generally perfectly normal (the cremaster reflex and the abdominal-wall reflex I have found in part normal, in part indistinct) the tendon reflexes are entirely absent. Next to the ataxia the extinction of the patellar reflex in especial is the most important and most constant symptom of the disease and also one that appears at a very early age. On the other hand, in contrast with tabes, reflex iridoplegia is practically never seen.

The vesical and rectal functions remain unaffected even in the advanced cases, again a very striking contrast to tabes. The psychical functions of the patients in the ordinary cases hardly suffer any material change. Yet a certain mental dulness is sometimes present in advanced cases.

Uncommon and Complicated Forms of Hereditary Ataxia.

As we have seen, Friedreich's disease is only an especial form of that great group of diseases which may be comprehended under the name of "hereditary system diseases." That form is to be designated the typical form of Friedreich's disease in which the clinical picture consists essentially in ataxia without disturbances of sensibility, absence of the patellar reflex, at a later time disturbances of speech and muscular paresis. But it is certainly not surprising if in a hered-

itary nervous disease nervous disturbances occasionally appear in other regions also, which likewise depend upon an abnormal hereditary predisposition of certain parts of the nervous system. There are numerous other hereditary nervous diseases besides Friedreich's disease, and it is consequently quite clear that Friedreich's disease might occasionally be combined with other symptom complexes, which in their turn are peculiar to certain other forms of hereditary nervous disease. Thus, for example, cases have been observed in which the symptom complex of Friedreich's disease was combined with progressive muscular atrophy (that typically hereditary or familial affection). The "peroneal type" of progressive muscular atrophy appears with especial frequency in conjunction with hereditary ataxia and then leads to the formation of a clubfoot, as already mentioned. The atrophy of the muscles about the shoulder, corresponding to "Erb's juvenile type," has also been observed. In other cases peculiar irritative motor phenomena, choreic movements and tremor, have been met with, symptoms which probably are related to the hereditary choreic and tremor affections. Marked spastic phenomena with abolition of the tendon reflexes, atrophy of the optic nerve, dementia, etc., have also occasionally been found in connection with other symptoms which correspond completely with Friedreich's disease. All these observations warn us against too schematic conceptions of Friedreich's ataxia. It is really much more strange that so many entirely similar "typical" cases actually occur, than that deviations from the type and transitions to other types of hereditary nervous affections are so frequently found. We believe then that if the subject be viewed from a general standpoint no especial difficulties will be presented by the variations in the symptoms of the individual cases.

Course and Prognosis.

As to the general course of Friedeich's disease we have little to add to that which has already been said. The further course of the affection is chronic like its beginnings, and a duration of ten to twenty years is not exceptional. Marked variations, especially improvement and remissions of the disease, hardly occur except perhaps slight transitory improvements, which generally are to be ascribed to more complete rest or better care and nourishment of the patients. We can speak more correctly, however, of temporary periods of arrest of the disease. At least I know of cases in which careful investigation detected no clearly perceptible change in the manifestations of the disease after intervals of one or two years. Nevertheless the course of the disease in general is steadily progres-

sive and incurable. The ataxia steadily increases and the paresis of the muscles becomes more and more marked. New disturbances (speech disturbances, mental weakness, etc.) make their appearance. The general nutrition and strength become more impaired.

The final fatal issue, which is often preceded by long illness, is the result in part of the general loss of strength, in part of secondary complications (accidental acute diseases, pulmonary tuberculosis, and the like).

Pathological Anatomy.

Our knowledge as to the pathological anatomy of Friedreich's disease is in many respects defective. New and thorough pathological investigations aided by good methods and pursued with reference to our present incomplete conceptions as to the anatomy and physiology of the nerve paths are urgently needed.

Relatively best known are the anatomical changes in the spinal cord. This has repeatedly been found as a whole remarkably small and thin (also the medulla oblongata). This condition depends perhaps in part upon a congenitally abnormal constitution, in part it is also probably the result of the loss of numerous systems of fibres and of the general inactivity. As another sign which points with the greatest probability to abnormal congenital conditions it may be mentioned here that congenital abnormalities of the central canal (syringomyelia, central gliosis) have been repeatedly observed.

The characteristic change of the spinal cord consists in a combined systematic degeneration of various fasciculi of fibres in the posterior and lateral columns. In the posterior columns the degeneration is widespread, as a rule certain marginal zones in the vicinity of the posterior cornua (which contain short paths) alone escaping. Not only Goll's but also Burdach's columns are much degenerated. Nothing is known with certainty as to the condition of the dorsomedian sacral fasciculus in the lumbar cord, which generally remains intact in tabes, but it seems to be unaffected likewise in Friedreich's disease. There are contradictory statements as to the condition of Lissauer's zone near the head of the posterior cornua. In some cases which were carefully examined, Lissauer's fibres were in the main well preserved, which is a decided contrast to their condition in tabes. The fibres which enter the gray posterior columns are much diminished and the posterior cornua are consequently distinctly lessened in size. Nothing is known as to the condition of their ganglion cells.

In the lateral columns there is constantly found marked degeneration of the direct cerebellar tracts and a generally slighter degenera-

tion of the lateral pyramidal tract. When the anterior pyramidal tract is present, this is also implicated. The so-called fasciculus of Gowers (anterolateral ascending tract) appears as a rule to be simultaneously affected. The gray anterior cornua and the other portions of the spinal cord remain normal. On the other hand, the cells of Clarke's columns from which the fibres of the direct cerebellar tracts arise are found to be atrophied.

The posterior roots are distinctly atrophied and the number of their fibres is diminished. The anterior roots are normal. As to the condition of the spinal ganglia nothing definite is as yet known, nor have we knowledge, which is greatly to be desired, of the condition of the peripheral nerves. The brain is essentially normal. Especially are there found no striking abnormalities in the cerebellum in genuine Friedreich's disease.

The changes, then, which may be at present accepted as certain, are marked degeneration of the fibres of the posterior columns and of the direct cerebellar tracts, and generally a slighter degeneration of the pyramidal tracts. It will be seen that these anatomical findings agree well in general with the two chief clinical symptoms of the disease. The ataxia, so far as it affects the trunk, certainly depends upon the disease of the posterior columns, perhaps also upon that of the direct cerebellar tracts. The paresis, later in development, is the result of the implication of the pyramidal tracts. The whole clinical course of the disease proves that the affection of the posterior columns always long precedes that of the pyramidal tracts. For symptoms due to the posterior columns alone are present for a long time (ataxia, absence of the patellar reflex), and it might be possible that, if a case of Friedreich's disease accidentally came to autopsy at a very early period, no degeneration of the pyramidal tracts would be found.

The fact is surprising and at the first glance almost unintelligible that in Friedreich's disease, notwithstanding the often very extensive disease of the posterior columns, disturbances of the sensibility of the skin and of the deeper tissues are practically entirely absent. This physiologically very important fact can only be explained provisionally by the assumption that the fibres devoted to the conduction of sensory impressions to the brain for the most part do not run through the posterior columns, but enter at once into the gray matter of the posterior cornua and then pursue their further course in the lateral columns. Attention is to be directed in this connection to the fact that Lissauer's zone is not implicated. The centripetal fibres of the posterior columns must consequently have chiefly coördinating functions. Just as remarkable is the absence of disturbances of the

bladder and rectum. With regard to this point, a careful comparison of the anatomical findings of tabes with those of Friedreich's disease might bring to light important facts.

We have no knowledge whatever of the anatomical basis of the other rarer symptoms of Friedreich's disease, especially the disturbances of speech and the nystagmus.

Diagnosis.

The diagnosis of a typical case of Friedreich's disease is not, as a rule, of the slightest difficulty to the experienced. The appearance of the symptoms in early youth, or perhaps in other members of the family, the ataxia of all the extremities and of the trunk with absence of patellar reflexes but with normal sensibility of the skin and the deeper parts—all these are such striking phenomena that they can hardly fail to be recognized. Friedreich's ataxia is distinguished from tabes dorsalis in especial by the absence of lancinating pains, of disturbances of sensibility, of bladder symptoms, and of reflex iridoplegia. I have never known any difficulties to arise in the differential diagnosis between tabes and hereditary ataxia.

Multiple sclerosis may sometimes require consideration in the diagnosis. The ataxia of the extremities together with absence of all disturbances of sensibility which occur in this disease may produce a clinical picture which at the first glance resembles Friedreich's disease. But the ataxia of the trunk, which is generally much more pronounced in the latter disease, and the absence of the tendon reflexes, in contrast with the spastic phenomena, which are hardly ever wanting in multiple sclerosis, make the distinction easy. The gait of Friedreich's disease is reeling-ataxic; the gait of patients with multiple sclerosis is spastic-ataxic. Certain symptoms are very often found in multiple sclerosis which are characteristic of it, but which are almost always absent in Friedreich's disease (epileptiform attacks and other cerebral symptoms, paralysis of the eye muscles, atrophy of the optic nerve, the early appearance of scanning speech, etc.).

It is of importance to discriminate Friedreich's disease from the so-called "hereditary cerebellar ataxia," which latter is chiefly due to a progressive atrophy of the cerebellum. In this there is also a reeling gait and a progressive ataxia of the extremities, sensibility and muscular force remaining normal. Cerebellar ataxia, which is also a familial disease, is distinguished from Friedreich's disease above all by the increase in the tendon reflexes. On the other hand, the fact must not be overlooked that the two affections, being both simply different manifestations of hereditary system disease, are of allied origin and

may therefore have certain relations to one another. Cerebellar and spinal hereditary ataxia on the one hand might be compared in a certain sense with spinal and neurotic muscular atrophy on the other. But with reference to cerebellar ataxia, further thorough anatomical investigations are much needed.

Treatment.

Since Friedreich's disease is due to an abnormal condition of the nervous system which cannot be changed, the possibility of influencing the disease therapeutically is unfortunately small. If the predisposition to the disease exists in a family an endeavor should be made on the one hand to strengthen the children who are not yet affected, and on the other to shield them from overexertion. Especially is excessive bodily exercise to be avoided.

When the disease is already present, general hygienic and dietetic measures are still of chief importance—nourishing food, good air, and avoidance of overexertion. Mild hydrotherapy (rubbing with cold water, lukewarm baths, douches, and the like) may also be tried, likewise electrical treatment at intervals—galvanization along the spinal column and galvanization and faradization of the muscles and nerves, especially if paresis is present.

A certain, but of course not great, benefit is sometimes obtained by the so-called "exercise treatment," *i.e.*, by the methodical exercise of the ataxic extremities by prescribed movements. Further particulars as to this method (which was introduced by Frenkel in the treatment of tabes dorsalis) are to be found in the article which treats of that disease.

It may be doubted whether drugs have any influence upon the course of Friedreich's disease. Nevertheless nitrate of silver, ergotin, arsenic, strychnine, etc., may be tried from time to time. For the symptomatic treatment various medicines (tonics, iron, preparations of quinine, etc.) are frequently employed.

In the last stages of the disease the physician will be obliged to confine himself to a symptomatic treatment of the individual symptoms and to securing the patient as good nursing as possible.

Hereditary Spastic Spinal Paralysis.

Hereditary spastic spinal paralysis is a disease which has become well known only in recent years. The number of clinical observations and, still more, the number of autopsies are therefore as yet far too small to permit at present a complete and comprehensive descrip-

tion of the disease. Nevertheless it is possible to affirm even now that it represents an especial form of hereditary system disease.

It is very important to distinguish at the outset congenital spastic paralysis from the family form of the disease which develops in later life. Congenital spastic paralysis is due to developmental abnormalities of the nervous system, for the most part an arrest of development of the chief motor tracts (so-called agenesis of the pyramidal tracts). This condition will not further concern us here.

The only form of spastic spinal paralysis which we shall discuss here develops slowly and gradually in individuals who were previously apparently healthy and had normal powers of locomotion. Without any demonstrable external cause a peculiar difficulty in moving the legs appears which may gradually increase to a very high degree. The age at which the first clinical manifestations make their appearance may vary greatly. But the fact is singular that the first symptoms are not infrequently detected in the third or fourth decades of life. But here as well as in Friedreich's disease the more careful the investigation the earlier will the beginnings of the affection be found to have appeared. It is certain that in a few cases the disease begins in early youth (in the tenth to the fifteenth year).

It generally occurs as a familial disease, and is sometimes also hereditary. This strange malady, then, attacks several brothers and sisters or several members of the same family belonging to different generations. It cannot yet be determined whether there is any noticeable difference in the liability of the two sexes. According to the observations at present available, the male sex seems to be more frequently attacked than the female sex.

Hereditary spastic spinal paralysis, like Friedreich's disease, may probably occur occasionally in quite isolated cases (in only one member of a family). But the observations are as yet insufficient to certainly decide this point.

Nor do we as yet know what the influence of accidental causative factors may be, although it will probably be found that they bear the same relation to this disease as they do to hereditary ataxia.

Symptoms and Course.

The symptoms almost always, it seems, appear first in the legs, the movements of which become more stiff and laborious. The gait gradually acquires all those peculiarities which characterize the true spastic gait. Examining the legs more closely, we find as the cardinal symptoms a heightened muscular tonus and an increase of the tendon reflexes. The muscles feel rigid, passive movement is made difficult

by instantaneous muscular resistance, and finally becomes almost impossible. The patellar reflex is marked; conspicuous fibrillary contractions are very easily produced. At the same time the nutrition of the muscles is quite normal, and sensibility is not affected. There is no ataxia, nor are there any disturbances of the bladder, of the mind, of the medulla oblongata, or of speech, and (upon this I wish to lay especial stress) the power of the muscles, as a rule, remains normal for a long time. The affection does not, therefore, lead at once to a paresis of the muscles but only to purely "spastic" phenomena without simultaneous diminution in muscular force. Hence, notwithstanding their spastic gait, the patients may sometimes walk for hours without fatigue. And even in the later stages, when the gait is becoming more and more labored, when finally the patients can advance only by small, stiff steps, the leg muscles under such tension that they are as "hard as boards," the feet almost "glued" to the ground, it is found on careful examination that the chief cause of this is the muscular rigidity, true muscular paresis being present only to a slight degree. The condition is found to differ from this just described only in the last stage of the disease or in a few special cases.

The course of the disease is always very slow. Years may pass, during which the patients are still able to work to a certain extent, since, aside from their singular stiff gait, they have really no symptoms. The arms especially retain completely for a long time their normal power of movement, and only a slight increase in the tendon reflexes is noticeable in them. Bladder disturbances almost never occur, or at least in only a slight degree. The nutrition and the electrical excitability of the rigid muscles remain normal. The sensibility finally manifests generally quite slight abnormalities, which can only be demonstrated in the feet or legs by close examination. These are for the most part only quite slight alterations of the sense of temperature, of touch, and the like.

The helplessness of the patients becomes extreme only when walking has become very difficult and when even in the sitting posture the muscular rigidity and the frequent clonic tremors (a simple result of the enormously increased tendon reflexes) have become annoying. No improvement in this condition can occur. Death ensues from some intercurrent disease or from general marasmus.

Pathological Anatomy.

So far as the as yet scanty anatomical facts permit an opinion, the chief anatomical lesion consists in a very slowly advancing degeneration of a tract in the vicinity of the lateral pyramidal tract. The chief fibres of the lateral pyramidal tract itself appear long to remain intact, to which the clinical fact also points that true paresis is so late in its appearance. The principal change must be undergone by those fasciculi or those fibres to which a reflex inhibitory function is to be ascribed, the loss of which consequently causes the characteristic increase of the muscle tonus and of the tendon reflexes. The degeneration in the vicinity of the lateral pyramidal tracts is most distinct in the lumbar cord, diminishing towards the cervical cord. How far upwards it may be followed further investigation must show. Besides the degeneration in the lateral pyramidal tracts a slight degeneration of Goll's columns and of the direct cerebellar tracts is also found. We do not yet know whether these changes are of any considerable clinical importance. The cells in the gray matter of the anterior cornua are normal.

Treatment.

As follows from what has been said, the treatment can only be symptomatic. Simple warm baths are of the most service. The effect of weak, stabile galvanic currents (cathode upon the spine, anode upon the muscles) may be tried. Little is to be expected of internal remedies (arsenic, bromine, etc.). The symptomatic treatment is governed by the special individual indications.

Secondary System Diseases.

We have finally to consider briefly the combined system diseases in the lateral and posterior columns of the spinal cord which are due to exogenous causes, especially syphilis, poisons, severe constitutional affections, and pernicious anæmia.

The Sequelæ of Syphilis.

A small number of observations makes it seem probable that nervous affections may develop as the result of syphilis in which, in contrast with tabes dorsalis, the degeneration is located at first and chiefly in the lateral columns (lateral pyramidal tracts) and attacks only to a slighter extent and at a later time parts of the posterior columns (columns of Goll) and the direct cerebellar tracts. A com-

plete picture of this form of postsyphilitic disease cannot be given as yet, but the condition is essentially a slowly developing spastic paraplegia of the legs without any considerable disturbances of sensibility and with generally only slight vesical disorders.

The recognition of this condition during the life of the patient is as yet very uncertain. The disease may be suspected from the etiology (previous syphilitic infection), the absence of heredity, and the on the whole somewhat more rapid course in contrast with the familial form of spastic spinal paralysis; on the other hand, the so-called syphilitic spastic spinal paralysis of Erb, which is due to syphilitic dorsal myelitis, is even more acute in its beginnings.

But much more knowledge must be acquired before we can give an accurate description of this form of combined system disease.

Toxic System Diseases.

The affections of the spinal cord in lathyrism and in pellagra, which have only recently been well studied, belong in part here. The careful anatomical investigations of Tuczek have proved with certainty the occurrence of combined system diseases in the spinal cord in the latter disease especially. In lathyrism and in pellagra the clinical spinal symptoms also appear essentially as a spastic pseudoparalysis (*i.e.*, impeded locomotion, increase of muscle tonus and of tendon reflexes) or spastic paraplegia.

Combined System Diseases in Severe Constitutional Affections, Especially Pernicious Anæmia.

In examining the spinal cord of patients who have died of pernicious anæmia, aside from diffuse changes and hemorrhages, fascicular changes are also found in the posterior and lateral columns which are to be regarded as systematic degenerations. The cause of these spinal changes is probably to be sought particularly in toxic influences which have developed in the body of the patient.

The clinical importance of these combined affections of the tracts of the spinal cord is not great, since the spinal symptoms, if noticed, are quite overshadowed by the severer manifestations of the principal disease. Nevertheless we should make it a rule in severe anæmia (pernicious anæmia, leukæmia, etc.) to examine into the condition of the nervous system. If unmistakable spinal symptoms (absence or exaggeration of the tendon reflexes, paresis of the legs, slight disturbances of sensibility, and the like) are found, we may infer with tolerable certainty the presence of the above-described anatomical changes in the spinal cord.

Besides the three groups of combined system diseases of exoge-

nous origin already mentioned, there may perhaps be other forms. But our knowledge of them is as yet so scanty that no positive statements can be made concerning them.

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PAIN.

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PAIN.

THE present contribution undertakes an examination of the phenomena of pain chiefly from the point of view of the science of psychology. The form and content of the presentation of the subject, however, have been determined in part by the circumstance that those for whom it is intended are members of the medical profession. Although my consideration of pain must depart somewhat from the plan that would have been adopted by the clinician, for whom pain exists chiefly as a symptom of disease, still the problem of the genesis of pain and of its relation to other mental conditions and to neural processes is common ground. Clinical evidence cannot be ignored by the psychologist, nor ought the physician to overlook the facts and theory of psychology. The attitude of psychology toward the nature and origin of pain as a mental state must influence, directly or indirectly, the consideration accorded it as a condition of the sentient human organism by neurologists, physiologists, and practitioners of medicine. The notion that pain is a "purely subjective condition," a "feeling," "a state of mind without any physical basis in the body," may modify the attitude of a physician even in treating the simpler ailments of mankind. How much more may we expect the attitude of the neurological clinician towards pain to modify his views of the localization and causation of a disorder which may present pain disturbances as the most important symptoms! Thus we find Starr treating an "hysterical pain" by hypnotic suggestion, and maintaining it to be "subconscious, subjective, mental, without any physical basis," an opinion which has its roots in "rational psychology," finds modern representation in Janet's well-considered theories of psychic automatisms, and runs riot in Myers' notion of hysteria as a disease of the subconscious self.

Psychology in truth has been far from unequivocal in its consideration of pain. Many instances could be cited where the same authority has now called it a sensation, while anon classing it, in the light of theoretical speculation, with the "subjective mental states" or "feelings." But on the whole, traditional psychology may be said to regard pain as a feeling—*i.e.*, a purely mental state or condition, with or, more frequently, without a physical basis in the nervous

system. That such speculative considerations are likely to modify the views of physicians is evidenced by the fact that White quotes Ziehen to the effect that "pain is not a sensation but a feeling-tone"; and Dana, to whose point of view I have had occasion to refer in an earlier article on this same subject, maintains that "psychologists seem to have come to the conclusion that pain is not a sensation but a form of feeling, and that in attempting to locate pain tracts we were pursuing a 'will o' the wisp.'" Another illustration of the influence of psychological theories is found in the phrenology of Gall and Spurzheim, whose errors are more directly traceable to a faulty psychology than to ignorance of anatomy and physiology.

The "new phrenology," if we may give that appellation to the modern theories of the cerebral localization of those functions of the nervous system that underlie consciousness, is likewise ultimately based upon current conceptions in psychology. The advance of cortical physiology will be furthered by the progress of psychological analysis, as the latter must also change in meeting newer conditions revealed by the neurohistologist. Whether it be the interpretation and location of "speech centres" or a "speech zone," or be the differentiation of the reflex from the conscious activities of the nervous system, conclusions arrived at by physiologists will be largely determined by the type of psychology that happens to have impressed itself upon the advocates of these respective physiological hypotheses. Considering the present state of psychology, its greatest service here would seem to be in supplying physiology with a working hypothesis for the explanation of its own data, carefully refraining from the imposition of speculative dicta, that may perchance put new difficulties in the way of an explanation of physiological processes. It is with a desire to avoid unnecessary and dogmatic statement of psychological opinion, and at the same time with the hope that I may succeed in making clear some essential analytic considerations from the psychologist's standpoint, that I now proceed to an examination of the mental significance of pain, and of its relation to physical stimuli of the environment and to physiological processes of the body.

The existence of a mental experience, pain for example, may be posited in one of two very different ways, according as it is "my" pain or the pain of "some one else." These I shall call the subjective and the objective methods. If the method be the subjective one, the investigator has immediate possession of the mental state under observation. Its existence, therefore, requires for him no further demonstration. But in studying the mental states of others, the investigator has to depend upon bodily movements as the expressive symbols of something unseen, unfelt, unexperienced. The activities

of the sense organ, such as the moving eye or the feeling hand, are then taken to be facts presumptive of the existence of accompanying sensations. The look of anguish on the face and the start of the body in surprise are differential indices of diverse mental conditions. The subjective method, however, does not leave the investigator alone with his sensations and feelings, for it carries with it the possibility of the investigator's observing his own bodily processes as these change with modifications of his mental content. In consequence, psychology has always been psychophysiological in reporting the relation between mental states and bodily processes or bodily structures. Thus the generally accepted definition of the sensation has been, not a subjective definition, but a psychophysiological one, namely: "A sensation is the mental effect or consequent or accompaniment of the excitation of a sense organ."

Furthermore, every subjective observer has also called into explanatory relation with the mental states that he is observing in himself the objects of the physical environment amid which he spontaneously guides his observed body and which he finds acting upon the body to produce those sensations. Thus he has always referred some of his mental states to the physical environment, giving psychology thereby a psychophysical bias or direction. The most fundamental distinctions of traditional psychology are psychophysical in nature, such for example as the tripartite division of all mental phenomena into cognition, feeling, and volition. As defined by traditional psychology, cognition is that state or process of mind in which the observer finds himself aware of the existence of external objects, the elements of cognition being sensations which are evoked in mind by the peripheral sense organs. Volition, on the other hand, is that state or process in which he observes himself directing the movements of his body. Feeling, finally, is conceived as a purely subjective state, not referable to either sense organs or muscles.

Beneath all the discussion of the nature of pain or the possibility of the existence of pain nerves there is found in the works of many psychologists an undercurrent of opinion that assumes the pain state to be a self-existent feeling, that is, a purely subjective condition having no reference to or causation in any body state whatever, central or peripheral. Modern theories of mental and neural parallelism, however, have necessarily modified this view of feeling so as to accord better with the psychophysiological postulate that every mental state or process has a correlated neural process. The views of Marshall aptly represent the modified traditional attitude of psychology; the pain state is regarded by this modern successor to Aristotle, Hamilton, and the English Associationists as the accompaniment of

modifications in the cerebral processes which underlie other mental states and which are therefore not referable to any specific or particular peripheral or central excitation.

A few words may be advantageously given to the relative value of the two methods, the subjective and the objective, as revealed by practical experience. If the investigator study pain in himself by the so-called subjective or introspective method, he has given him as original data the "pain," the object which caused it (a hot stove, for instance), the hand which had come in contact with it, and the contractions of his own face or the revulsive movements of his own body. The objective method, on the other hand, forces the investigator to assume that which is the object of his study, the pain state, from the contact of some one else's body with the stove, and from the contortions of a face or the revulsive movements of the whole body. This assumption of a definite mental state is one attended with great difficulty, and with danger of error, even in the higher animals and in children. It is only by a considerable stretch of analogy, which oftentimes is carried beyond the breaking point, that the existence of pain states can be inferred in animals whose brains and bodies differ markedly from ours. It must be added, however, that in the adult human being we are assisted by the articulate words of the subject, who may report upon the presence or absence of pain and give, with more or less exactness, a verbal description of its qualitative and other subjective peculiarities. It is true that spoken words are psychophysically only the coördinate movements of an articulatory mechanism producing an expressive externalization of a mental state, but our attitude towards the speaking human being places him, under these conditions, in the position of the subjective psychologist, in virtue of which we take his reply as a peculiarly singificant evidence of the subject's mental experience. The two methods, therefore, in practice approach one another in their results. Nevertheless it will depend largely upon the natural and untrained powers of introspection and verbal expression possessed by a given individual, whether the verbal report of his subjective state or his behavior under the action of a determinable stimulation furnishes the better evidence of the actual mental conditions. Physicans do not place much confidence in the patient's statement of the quality or character of a pain, largely because patients have not words, nor experience to prompt the words, in which to describe their pains. Yet old sufferers will often describe their pains with great picturesqueness. In this field careful studies have been made by Henschell, W. H. Thomson, and others. The description of pain by a patient seems to be directly proportional to (1) liveliness of the imagination (the

Irish and Jews are said to describe pain most vividly), (2) vocabulary, and (3) experience. The truth of the description, however, depends upon other factors than these, many of them not directly ascertainable; but among them the chief are perhaps acquired skill in introspection and the absence of a tendency to hallucination and illusion.

Few attempts have been made up to the present time to study pain phenomena by the purely objective method. An exceptional study in this field is the examination and classification of the phenomena of pain with reference to the patient's behavior undertaken by W. H. Thomson. He describes and classifies the gestures that are characteristic of the different varieties of pain, noting their significance for the particular affection that is the causative agent of the pain. He maintains that gestures often afford truer indications than does language, because the verbal descriptions obtained may be so extremely indefinite. Some of his descriptions are so suggestive in pointing out new possibilities for refinement in the observation of the bodily movements accompanying pain that a short selection from his article may advantageously be quoted here. For the purpose of a further classification according to gestures, pains are divided into the following groups: (1) Pains due to inflammation; (2) pains due to pressure; (3) pains due to stretching; (4) neuralgic pains; (5) subjective pains; (6) reflex cutaneous pains. Many objections to this classification will arise in mind—for example, stretching and pressure pains are identical in physical causation, subjective pains are questionable in terminology, the classification is not based upon any one system throughout, etc. But the classification is probably only intended to be a roughly convenient one, and moreover it is the description that interests chiefly, as an illustration of a valuable method that needs further development.

With inflammatory pains we are told the patient "avoids touching the painful part, or he approaches it in a very respectful way." "Thus, with an arthritis, his hand passes over the joint in a hovering fashion. If deeper seated, the gestures are often expressive of the varying kind and distribution, according to the texture inflamed. Thus the diffused soreness of a mucous membrane inflammation causes the gesture of bronchitis to be made, with the whole hand laid on the sternum, and then passed over and across the chest. A similar movement of the hand across the abdomen never means a peritonitis, but a catarrhal intestinal inflammation. With pleurisy, on the other hand, the tips of the straightened fingers are used to indicate the stabbing nature of the pain. In peritonitis also the tips of the fingers are used, but brought down with much more caution than in pleurisy.

In the localized pain of commencing appendicitis, the open hand is used as with an inflamed joint."

To these pains are contrasted those produced by pressure, tumors, abscesses, etc., or cramps, in which no apprehension is manifested in touching or in moving the part. The locality first touched is of importance to note, because if the patient starts from the same place, each time his hand moves over the painful area, his movements may be considered expressive of the extension or radiation of the pain from the original focus. Stretching pains contrast with inflammatory pains by the forcible grasp or pressure which the patient makes on the abdomen, while the characteristic radiation may signify the causation as due to biliary or renal colic or cramps, etc. Neuralgic pains are indicated by a frictional movement of the hand, with firm pressure. The shifting character of the neuralgic pain as shown by the gestures of the subject enable one to distinguish these from the more fixed pressure pain of tumors. The darting pains of tabes are often most graphically described by the gestures. Subjective pains, as in hysteria, are characterized by their greater number and variety, and by the inconsistency of the gestures with the often highly wrought description of them by the patient. The character of referred pains can always be proved by the fact that the patient has no objection either to movement or pressure of the part.

Whether our method of approaching the phenomena of pain be the subjective or the objective, pain may be considered as a state of mind from one of three points of view: (1) The psychical, which views the pain as a mental phenomenon in its relations to other mental states only; (2) the psychophysical, which views it in relation to the external physical causes or stimuli of the pain; and (3) the psychophysiological, which is concerned with the pain in relation to the associated processes of the body. It is often difficult to keep these lines of treatment distinct, but it is necessary for clearness that they should be held as far apart as possible. I shall now proceed to consider pain from each of these three standpoints in succession.

The Psychological Problem.

The psychical, or, as more commonly called, the introspective line of treatment must comprise (*a*) an examination of the different qualities of pain, (*b*) a consideration of the relative simplicity or complexity of the pain as a mental state, and (*c*) a presentation of the common characteristics of pain and of certain other mental states which show resemblances to pain.

Pains have been described as dull, throbbing, sharp, massive,

grinding, shooting, burning, chilling, boring, griping, shivering, creepy, itching, and formicating. Potain reports that a physician in Trajan's time held that there were thirteen different kinds of pain, while Halmemann, another early physician, distinguished as many as seventy-five varieties. Most of these supposed varieties of pain cannot be looked upon as variations in pain quality. They probably arise from the simultaneous association of the pain with other mental qualities or elements. Chilly, burning, dull, sharp, massive, grinding, shooting, boring—these descriptive terms all refer to other sensations which are associated with the primary pain sensation. To take another group of composite sensations—those of taste—we find the sensations of taste proper to be always closely associated with sensations of smell, touch, heat, cold, and probably also with muscular sensations, as in bitter tastes. The loss of the sense of smell often apparently abrogates the sense of taste, because we can recognize so few substances placed in the mouth by the four pure taste qualities of salt, sour, sweet, and bitter. Tickling, shivering, creeping, shooting, formicating, intermittent, itching, paroxysmal, darting, or lancinating pains are distinguished from one another and have received distinctive appellations, not through inherent qualitative differences in the pain, but through variations in the duration of the pain, its mode of arousal by the physical stimulus, or its distribution throughout the body. Each one of these distinguishably different group pains ought to be made in itself the object of careful examination by psychologist and physiologist, if we are to have what we do not now possess, a satisfactory analysis of these complex sensations. Although we may not be able to designate the particular modifications of physical, physiological, and mental processes that are the basis of these variations in pain quality, we are still able to posit as a fact of introspective analysis that a pain, as pain, is always the same invariable mental experience no matter how closely it may be associated with other sensations.

Certain mental conditions are connected with pain in such an intimate way that they must be given special consideration at this point. These are: 1. The feeling of the agreeable and disagreeable; 2. The feeling or sensation of intolerableness or unendurableness; 3. The pains of inhibited motor process; either in the general sense, as those due to an insufficiency of bodily exercise, or more specifically, the pain or distress due to an interruption of some motor paroxysmal process which is on the point of taking place—for example, an interrupted gape or sneeze, the initial stage of nausea, and probably also the pain of restrained sexual desire; 4. Vertigo; 5. Pains of overexertion or fatigue

It is maintained by some authorities that pain is only an extreme case of disagreeableness, and pleasure merely the intensest agreeableness. Agreeableness or pleasure and disagreeableness or pain, in this view, are regarded as an "affective tone," a "mental color," an "attribute" or "quality" of some sensation or other specific mental content—this "affective tone" having two qualitatively different phases, the agreeable and the disagreeable, each presented with variations of intensity, in all degrees from indifference to the extremes of pleasure or pain. We are asked to think of this affective quality, which according to Marshall, the latest exponent of this view, is "a primary quale affecting all presentation," somewhat after the manner in which we grasp the notion of intensity as being common to all sensation. To this subjective modality of sensation psychologists generally give the name "feeling-tone," for which awkward foreign expression Marshall suggests as an English equivalent the hyphened compound word "pleasure-pain." In such authors as Külpe, Ziehen, Höfding, Lehman, Ward, and Marshall, feeling-tone embraces the agreeable and disagreeable elements in all sensation, and includes as manifestations of this single affective variable extremes of both pain and pleasure. The painful or pleasurable phase of feeling-tone is regarded as dependent directly upon the intensity of the sensation. If any given sensation, therefore, could be presented in mind in all possible intensities, it would be capable of arousing the extremes of pleasure and pain together with all pleasure and pain intensities between these and indifference. The quale theory presupposes that a bitter taste of adequate intensity must give a pleasure equal in degree to that of the satisfaction of the sexual instinct, while the odor of violets if sufficiently intense must arouse a pain as decided as the agony of angina pectoris. This *reductio ad absurdum* presents a real difficulty of the feeling-tone hypothesis, which is equalled by two others that amount to a contradiction of the evidence of introspective analysis. The first of these is found in the fact that many sensations are not presented in consciousness with either pleasure or pain; they are simply indifferent, no matter what their intensity may be. The quale theorist is compelled to produce the phantom of a feeling-tone that excites no consciousness, though still present, in order to explain this difficulty. Again, this theory maintains that no pain ever comes into consciousness except as a constituent part of some sensation or other mental state. In the words of Bradley, "we never have pleasure or pain; it is not a pleasure but something pleasant that is experienced." Ward complains that "physical pain is wrongly classified among bodily sensations, because one word, pain, is used for certain organic sensa-

tions and for a purely subjective state." Lehman maintains that the "feeling whether of pleasure or of pain never occurs apart from a sensation however weak. And in every case where such feeling is supposed to have been observed, the sensational element has merely been overlooked." Külpe explains this fact in the following words: "Since the presence of a strong feeling conceals the quality of its concomitant sensation, the analysis of a class of sensations" (peripheral pain and the pleasure of sex) "which hardly ever make their appearance except under this condition will obviously be exceedingly difficult." Thus does the quale theory carry with it the necessity of assuming the existence of some sensation to which pain may be attached, even in those extreme pains which apparently usurp consciousness to the exclusion of all other content. This view is somewhat like regarding the whole man as belonging to the tapeworm concealed within his vitals or as the property of the shadow that he casts upon the ground.

A view more in accord with the evidence of introspective analysis looks upon pain as a simple unanalyzable and undifferentiable content of the mind whenever it may be found existing within an individual consciousness. Fechner maintains that "pain and pleasure, when considered apart from all accompanying conditions, are simple, that is, they are not further analyzable as contents of our mind. They do not, however, exist so abstractly in reality as they may be apprehended by our faculty of abstraction, but rather as coexisting states or resultants, perhaps functions, of other mental contents, to which they give and from which they receive character." Wundt, also, in criticising Lehman's theory, maintains that the "use of the word pain for a disagreeable feeling of every sort has led to the notion that pain is not a differentiated sense quality." Münsterberg, Nichols, James, Strong, and many other psychologists, regard pain as a simple specific sensation. From this view there is no escape so long as we use the term sensation with a purely mental reference. Pain is certainly presented in consciousness with the distinctness, difference, vividness, and isolation that characterize simple sensations. If we add to this fact of mental differentiation other criteria, as we may do inferentially, even by the use of the term sensation (because this term implies, as I have pointed out above, that a sensation is the resultant of the stimulation of a sense organ), then objections may arise to calling pain a sensation. Such objections, however, will be based upon considerations that emphasize the psychophysiological rather than the strictly psychical characteristics of sensation. It is true that the tendency of psychophysiology to-day, whether rightly or wrongly I shall not inquire, is to demand for every differentiated

sensation a specific structure whose function gives rise to the specific simple mental content. If from the standpoint of introspective analysis, therefore, pain in common with all sensation is a simple mental content, we are presumably justified in looking for an anatomical substrate in a peripheral sense organ, a conduction tract in the peripheral and central nervous system, and a localized centre in the cortex of the cerebral hemispheres.

Having thus disposed of pain as a distinctive mental quality, we shall consider its intensity and some further characteristics that it presents in relation to the agreeable and disagreeable and to the feeling of intolerableness. Of pain intensity we shall say a word in a subsequent section on the psychophysical problem. The relations of the pain state to the agreeable, I can best discuss by a continuation of the consideration of the nature of feeling-tone. It is to be remarked that we may regard pain as a sensation and yet hold to the quale theory, so far as the feeling-tone of agreeable and disagreeable in respect of sensation in general is concerned. This is Wundt's notion, which leads him to treat feeling-tone in its phases of agreeableness and disagreeableness as the accompaniment of all sensation, even of that of pain. He would perhaps agree with James in the latter's statement: "I think we find here that the distinction has to be made between the primary consciousness of the pain's intrinsic quality and the consciousness of the degree of its intolerability" (Wundt would say disagreeableness) "which is a secondary affair simply connected with reflex organic irradiation." Münsterberg maintains that the pleasure of the sexual function (*Wollust*) and pain (*Schmerz*) are not the extremes of agreeableness and disagreeableness (Lust and Unlust), but are sensation contents which regularly arouse strong agreeable and disagreeable feelings; he further believes that "even if the explanation of pain and related sensations as due to peculiar trophic nerve processes or to specific nerves of feeling is correct, still nothing is advanced thereby towards an explanation of the feelings of agreeableness and disagreeableness." Thus these authorities appear to agree in maintaining that the disagreeableness of pain is not to be explained by reference to the pain content as such, but to associated bodily conditions or sensations which, in the terminology of Wundt, receives the name of "feeling-tone."

This leads me on to an examination of the nature of feeling-tone. Wundt offers no satisfactory explanation of its nature, accepting it as an ultimate mental fact to be corollated with varying intensities of the sensation. James suggests that intolerability may be connected with reflex organic irradiation. Thus the violent re-

traction of the head from ammonia compels us to speak of the odor of ammonia as intolerable or unpleasant, but this is a psychological misapplication of terms. The ammonia may be unpleasant as well as odorous, but the sensation of the odor in itself is neither pleasing nor displeasing. The substance ammonia acts reflexly upon our nervous organization to produce movements of retraction, and these movements themselves give rise to the feeling-tone of disagreeableness. Onions may give rise to what we should call a disagreeable odor and may at the same time act upon the tear ducts to cause a secretion of tears, but strictly we are no more justified in calling the odor disagreeable than we should be in calling it tearful. It happens that the onion produces tears and disagreeableness at the same time that it produces the specific odor sensation. An insect on the skin is a very harmless thing in itself, but it requires all our endurance to watch the harmless fly or spider walk about over the surface of the skin, because this stimulus awakens powerful reflexes to remove the stimulating agent. Because of these reflexes, we appropriately regard the insect as intolerable, but wrongly believe that the sensation of touch from the insect is also intolerable, forgetting that the one object, the insect, is both intolerable and touching. A drop of acid arouses as inoffensive a sensation of touch as does a drop of water; but the acid does more to us than does the water; it burns the skin, provoking sensations of burning and of pain and arousing violent reflex movements of retraction. Agreeableness and disagreeableness, which seem to merge into pain, can therefore be explained by reference to bodily conditions which the stimulus provokes in addition to the physiological processes that evoke the specific sensation. Thus a stimulus acts also upon the internal organs of the trunk, for instance on the stomach and on the involuntary musculature of the intestines; it acts on the vasomotor system producing constriction; and finally it may act upon the peripheral muscles through the excitation of reflex centres in cord and brain.

The sensory ground work for feeling-tone may be found in any one or all of these reflex bodily excitations. It may be placed in the organs of the trunk (James), which seems to explain best the disagreeableness that comes from concomitant feelings of suffocation, fear, etc.; or in the vascular system (Lange), which may explain chilly and creepy and possibly formicating and other pains; or in the reflex contractions of the striped muscles of the periphery and trunk. It is also a possible hypothesis that the disagreeableness of a sensation may be due to the voluntary or involuntary inhibition of a reflex movement strongly excited. The disagreeableness of the walking insect, for example, may be referred to the fact that our bodies cannot

move quickly enough to satisfy the demand of the stimulus for motor tracts to the muscles productive of retraction. On this basis, disagreeableness would be an unsatisfied muscular craving or desire, to be placed with the malaise or discomfort due to want of exercise, unsatisfied sexual desire, an inhibited sneeze or gape, and so on. Münsterberg's theory, on the other hand, relates the agreeable and the disagreeable feeling-tone to definite antagonistic motor processes. The results of certain experiments furnish evidence (not highly satisfactory, however) that all pleasurable states of consciousness are accompanied by bodily movements of extension and all painful states by bodily movements of flexion. These movements may be very slight and need not be referred to the extremities only. Münsterberg's conclusion is that the feeling of agreeableness is the mental accompaniment of reflexly produced movements of extension, and the feeling of disagreeableness of reflexly produced movements of flexion. A stimulus, such as a very hot object, through the medium of an afferent nerve impulse, excites certain areas of the central nervous system concomitant with which is a sensation of heat; the central excitation either in the cortex or in centres lower down reflexly produces innervation of the musculature involved in flexion, which in turn serves as a stimulus for a secondary afferent impulse, producing a cortical excitation whose mental accompaniment is the feeling of disagreeableness. Analogously, the mental resultant of reflexly excited movements of extension is the feeling of agreeableness. This theory is maintained to be in harmony with the physiological observation that even when the original motor impulse passes from the nerve centre, there can be produced either peripheral flexion or extension, according to the intensity, duration, and extent of the central excitation. As the central motor impulse is normally the effect of centrifugal stimulation, it appears fair to assume that the primary sense impulse will, in accordance with its intensity, extensity, and duration, excite in the central substance the motor impulse towards either flexion or extension. In other words, the stimulus, which with moderate strength and short duration reflexly excites extension, will with greater intensity or longer duration produce flexion.

Although Münsterberg's theory does no great violence to the evidences of biology, physiology, and psychology, we cannot regard this interesting piece of speculation as final. These various hypotheses of the psychophysiological processes of feeling-tone, however, are undoubtedly making the nature of pleasure and pain more intelligible. Contradictory as they appear to be on the surface, all of them are in direct extension of the postulate of modern psychology, that every modification of consciousness may be viewed as the con-

comitant of some physiological process which is either of peripheral or of central origin, whether this be directly, associately, or reflexly stimulated.

The Psychophysical Problem.

I shall now proceed to consider the relation of pain to the physical stimulus—that is, the extracorporeal stimulus of origin. Much has been made of the fact that pain has no specific stimulus with which it can be corollated, as have sensations of color and of sound. In the latter group of sensations—that of sound sensations produced by atmospheric vibrations—we find the clearest cases for the relating of sensational quality and intensity with specific differences in the physical stimulus. A sound varies in pitch as the number of vibrations of the stimulus increases or decreases. It varies in intensity as the stimulus of sound is removed from the ear, thereby diminishing the amplitude of vibration of the layer of air next to the tympanic membrane of the ear. It is commonly maintained that every sensation has intensity and quality, both of which are referable to differences in the stimulus. The quality of a color sensation has been assumed to be due to the number of vibrations of ether, but modern psychology has rejected the notion that color sensations have intensity, despite the popular use of that term in describing visual sensations. I cannot enter into a discussion of the reasons for this; suffice it to say that psychology to-day looks upon all color sensations as differing from one another in quality only; the physical stimulus may differ in intensity, but intensity differences in the stimulus provoke qualitative differences in sensation. For further proof of this the reader must be referred to the works of Külpe or Wundt. Every sensation therefore is not necessarily a composite of quality and intensity.

Another illustration to show that quality and intensity of the stimulus are not always correlated respectively with quality or intensity of sensation may be given from the group of dermal sensations. A minimal pressure stimulus of 1 or 2 gm. will give a sensation of touch; greater intensity of the same stimulus ranging from 20 gm. to 15 kgm. will give a sensation of pressure; 5 kgm. to 15 kgm. will give both pressure and pain, while maximal stimuli above 15 kgm. give rise to pain only. This at least is the evidence obtained from tests conducted by me on a large number of persons. There are, therefore, three differentiated qualities, touch, pressure, and pain, each with an intensity range of its own, and each referable not to qualitative but to intensive differences in the physical stimulus. It is a confusion in thought, due to the fact that the same pressure stimulus acts upon us

to produce all three qualities, which makes us think that a common quality in sensation must also be present, and that a sensation of pressure is only an intense sensation of touch or that a pain from a pressure stimulus is a painfully intense pressure sensation. The same confusion leads to a view that regards a drop of acid on the skin as the cause of a painful or burning touch sensation, whereas the touch sensation as such is indifferent. It is the acid, the physical stimulus, that is touching and burning and painful. Intense pressure stimuli provoke pain sensations; so do intense heat and cold stimuli; so also does the stimulus of tickling, which is an intermittent touch stimulus of small intensity. Apparently all stimuli that can act upon the body at all are capable of giving pain—mechanical, chemical, thermal, and electrical stimuli. Pain, therefore, has no specific stimulus unless we regard the upper range of intensity of general stimuli as the specific stimulus of pain. But in any event, the fact that pain has no specific stimulus does not militate against its being a sensation. Other sensory qualities have been cited which are referable to intensity changes in the stimulus. The effects of intense stimuli in producing pain are better referable by way of explanation to the organism, especially to simultaneous action upon other nerve tracts, than to the specific nerve tract in question.

Similarly the effect of a temperature stimulus depends upon the temperature of the body—the physiological zero point. The same temperature stimulus may produce either a sensation of heat or of cold, depending upon the condition of the part acted upon. Nevertheless, we regard heat and cold as specific sensations despite their very shifting character relatively to the physiological zero point of the body. Although we may grant that pain can be aroused by a visual or an auditory stimulus, it seems to me questionable whether the irritation of either the optic or auditory nerve is itself capable of arousing pain; certainly the pains that are most frequently associated with the exercise of the functions of vision and audition are not due to optic or acoustic irritation but to the concomitant irritation of branches of the fifth nerve. Consequently, if a sound stimulus produces pain, it seems to me more likely that this is due to the action of the sound wave upon the tympanic membrane or upon other strictly dermal tissues or connected nerve fibres, than to the excitation of the specific nerve of hearing; similarly, a light wave is painful because of its action upon the conjunctiva. Certainly retinitis may run its course without pain, whereas all inflammatory processes of the conjunctiva and of the lid produce intense photophobia. The auditophobia and photophobia of meningitis also are scarcely ascribable to the nerves of audition and vision.

Many pains, such as those of fatigue, are not referable to extra-corporeal stimuli at all, but to the muscles. The stimulus here is probably a chemical one, the resultant of processes of dissociation in muscular tissue, this product acting as an irritant poison on the sensory nerve. The pains of muscular origin due to overuse have had an important influence upon theories of pain. Pains due to irritation of the viscera are probably also evoked by chemical stimuli, as in the pains of hunger and thirst, although some of them may be due to the involuntary contraction of the unstriped muscles of the intestines and perhaps also of the vascular system. But whether the stimulus be internal or external, it is not painful when at the physiological zero point, but becomes painful when excessive or when causing excessive functioning of a bodily organ.

It may be of interest to point out in this connection, two different lines of speculation of some importance to psychology, that have taken their origin from a formulation of the psychophysical setting of pain as regards its physical stimulus. The first of these is what may be called the economic or biologic theory, based upon considerations of the protective value of pain or of its contrasting pleasure to the human organism, particularly as these are found to be stimuli to diverse actions, pain warning against dangerous stimulation and causing the organism to avoid such stimuli, pleasure impelling to the search for serviceable stimuli. Economics as a science bases itself upon the conscious choice of pleasure. As stated by Patten, the "theory of utility is merely a conscious reckoning of our pleasures and pains. Passions and strong feelings are best controlled by analysis of our pleasures and pains into their parts so that we can determine the degree of each feeling and give to it a proper weight."

Psychologists have not been wanting who in the same way have considered pain and pleasure as the primary phenomena of consciousness or the primary determinants of the will. Spencer has made pain-avoiding and pleasure-getting, in relation to the dangers and useful objects of the environment respectively, a biological principle in psychology and sociology. Brentano calls pain and pleasure one with the fact of the will, while Meynert maintains that "the simplest theory will always be that in the most complicated, enigmatic, and incomprehensible actions of man, the guiding motive is the avoidance of the greater pain." Important as pain is in determining action, the facts seem to justify James' opinion that other conditions than pain cause bodily movements both in the region of conscious choice and in that of instinctive or reflex action, and many have pointed out that the reaction of the organism with pleasure and pain

is not always associated with the danger or usefulness of the stimulus to the organism.

Another line of speculation has taken the partial view of pain as this is found related to intense stimuli in connection with the over-exertion of muscles, and has developed theories which have been presented in various forms from the time of Aristotle to that of Marshall. Marshall has given the latest formulation of this theory, maintaining that the activity of the bodily organ of any sensation content "if efficient is pleasurable, if inefficient, painful." In other words, pain is experienced whenever the physiological reaction which determines a specific sensation is so related to the supply of nutriment of the functioning bodily structures that the energy involved in the reaction to the stimulus is less in amount than the energy of the stimulus. Neither this theory of Marshall, nor similar theories that have preceded it, throw much light on the problem of pain, nor can any refinement of speculation concerning energy conditions in the brain be expected to assist the explanation of the cortical substrate of pain, so long as these theories base themselves solely or primarily upon an observation of the excessive exercise of muscles and sense organs. Although pain is dependent upon intense stimuli which are generally dangerous to the well-being of the organism and which generally provoke excessive bodily reactions, these relations are far from invariable and do not furnish a satisfactory basis for theories of the nature and origin of pain. Such theories are strictly of psychophysical origin, and, in deducing physiological hypotheses from observations of the psychophysiology of gross bodily organs, they ignore important psychophysiological conditions that have been brought to light by the newer investigations of histology and neuropathology.

The Psychophysiological Problem.

Attention has already been given to the fact that traditional psychology looks upon differences in sensation as referable to different sense organs. The so-called "five senses" is a traditional classification of the peripheral agents of perception, that had some significance when physiology concerned itself chiefly with the processes of macroscopic organs. It has little value at the present time, except that of convenience in referring to the different groups of sensory structures. The theory of specific sense energies took its origin in part from this reference of a difference in sensation to a difference in sense organ, but in part also it is derivative from the investigations and theories of Müller, Purkinje, Goethe, and others as to the nature of phantasies, illusions, and hallucinations. In these early formulations of

the theory of specific energy, originated the tendency of to-day in psychology to refer memory, imagination, and ideation to the same brain organ that mediates sensation and perception. Helmholz is said to have followed a suggestion of Natanson in formulating the dictum of the theory of specific energy that no organ can exercise two or more functions different in kind. There must be a single response with a specific sensation to every sort of stimulation of the organ. This theory is rightly or wrongly accepted at the present day as a working hypothesis in the psychophysiology of sensation, and has inaugurated a search for physiologically and anatomically differentiated sense organs, peripheral nerves, nerve tracts and centres of the cerebral cortex, as the basis of specific differences in sensation. The theory of the specific energy of the total sense organ first found expression in a dogma that later investigation proved to be untenable. This was the theory that every nerve fibre was capable of one form of nerve excitation and no other. The search for peripheral pain nerves, however, still continues, although this earlier interpretation of the specific energy of nerve fibre has been abandoned.

The study of cortical physiology is turning attention from the peripheral to the central termination of the sensory tract. It may be said that the accepted belief is that every distinguishably different sensation has a locally differentiated cortical cell or group of cells as its anatomical basis, and that such central neural substrate can function with its specific sensation and no other. Wundt is one of the few psychophysiologists who maintain that specific sense energy may be a differential function of one and the same peripheral, if not central, organ. These are two contrasting points of view, and I shall now proceed to inquire to what extent specific differentiation in both structure and function, or differentiation in function alone, can be correlated with pain sensation. For this purpose I shall present the various parts of the discussion in the following order: (1) the peripheral sense organ; (2) the peripheral nerve or peripheral sensory neuron; (3) the conducting neurons of the spinal cord; (4) the conducting neurons of the brain; (5) the cortical neurons.

The Peripheral Sense Organ or Specialized Nerve Ending.

Goldscheider was among the first to call attention to the interrupted distribution of heat, cold, pressure, and pain spots over the surface of the body. Corroborated by later investigations, his results demonstrate that there are functionally specific sensory spots on the skin which are sensitive to pain stimuli, that is, respond to stimulation with a pain psychosis, and thereby differentiate themselves from

other parts of the skin. This fact would seem to compel an assumption of the existence of a specialized sensory cell or of a specialized nerve terminal for pain.

Later results, also, including those of Goldscheider, von Frey, Nagel, Dessoir, Singer, and others, despite what is oftentimes conflicting testimony, demonstrate with sufficient clearness and satisfactoriness the specific pain function of localized areas or "spots" of the skin. It has not, however, been possible to prove satisfactorily that the pain spot reacts with pain only to all forms of stimulation; but, in Wundt's opinion, this is equally true for cold, heat, and pressure spots. Despite the specific physiological character of the pain spots, the modifications in pain production due to stimulation are such that it cannot as yet be maintained that this functional differentiation is dependent upon a difference in structure.

There is no anatomical or pathological evidence of a direct sort demonstrating a terminal organ of pain, although evidence, coming from many sources, is abounding to show a marked functional differentiation in peripheral sensibility to pain over different areas of the skin under normal conditions and over the same areas under pathological conditions. For example, ice in the hand will produce cold and pain, while in the stomach it excites pain only. Local or peripheral anæsthetics will cause a loss of pain without the absence of other dermal sensations. Touch heat, cold, pressure, pain, and the deeper sensations composed of muscle, joint, and tendon sensations may, in lesions of central origin, as will be shown later, be lost independently over large areas of the body or over circumscribed regions. Within the region of anæsthesia, the loss of these different sense qualities is variable. Pain characteristically is most easily lost. Heat and cold follow next, while touch is seldom lost and the kinæsthetic sensations almost never. Nearly every possible combination of sensation losses has been reported in cases of spinal lesion. These facts taken together clearly demonstrate that the terminals of some or all dermal nerves function specifically with pain production and furnish almost sufficient evidence to warrant an assumption of the existence of a peripheral end organ of pain. Whether such assumption is justified or not remains a matter of opinion; in any event, the case for a specific end organ of pain is as well made out as for that of the other dermal sensations.

The Peripheral Nerve Fibre or Peripheral Sensory Neuron.

There is much evidence pointing to a specific pain function of one or all of the peripheral sensory neurons. Although the experiments of Goldscheider, Blix, and Nothnagel may not be able to show more

than three different peripheral paths even at the nerve roots, and thus there is little or no anatomical evidence pointing unequivocally to the existence of four different peripheral nerves nor even of three; still, pathological results are clear in demonstrating the same functional modification that the results of experimentation on localized pain spots, referred to in the previous section, have advanced.

It is remarkable with what exactness disturbances in pain sense can be localized on the periphery. The most careful examination that has yet been given of restricted or elective sensory paralysis in localized areas is that of Llewelles F. Barker, the results of whose examination of an anæsthetic area of his own arm are conclusive. His case is described as a circumscribed unilateral and elective sensory paralysis. For nine or ten years, the investigator had had along the inner middle side of the left arm radiating pains which were prone to appear on standing for a long time or after a long walk, especially when overtired or a little run down in health. Sensations of numbness and formication had been noticeable and were referred to the same area and also to the little finger and ulnar side of the forefinger. The pains could be stopped by lying down. There had been no other symptoms to modify this clinical picture. The last cervical vertebra exhibited in front, where it could be seen, a process firmly attached to it, which is described as a "cervical rib." Along the affected area were found certain areas of anæsthesia and analgesia, whose limits could be determined with tolerable accuracy by quick methods. This was done for cold, heat, pressure, and pain. The different sense points and their threshold stimuli were then carefully determined by exact methods of localization and measurement. The arm was divided into a number of sections according to the course of the visible cutaneous veins and also with regard to certain irregularities. Gelatin paper was laid on the arm and a copy made in position. For each quality of sensation a special plaster cast was made, upon which were traced the sensitive spots exactly as found upon the arm. The disturbance was found to be limited to the region of the nervi cutanei brachii et antebrachii mediales of the left arm. Within this area warmth, cold, pressure, touch, and tickle sensation were absent. Pain was present on superficial examination, but, with accurate testing, some diminution in the number of pain points could be demonstrated.

On the flexor surface, passing from the radial to the ulnar half, the sense points for warmth, cold, and pressure, which on the radial side were present in normal numbers, suddenly ceased when advancing beyond the anæsthetic border. In passing over the ulnar margin of the forearm to the extensor surface, single sense points here and

there again appeared, increasing gradually in numbers towards the radial margin, but not being present in normal numbers until the margin was nearly reached.

The limits described showed variations in detail for different sense qualities. They followed the same rule as on the normal skin, pressure being greatest in number, cold and warmth following in diminishing number. Single sense points or groups of points of one kind were to be found located in places where none of the other sense qualities was represented.

Sense points of the partially anæsthetic area, whether these were isolated or mixed with others, as well as points on the border of the normal and anæsthetic skin reacted to normal threshold stimuli. This could be positively stated for the pressure and pain points from numerous determinations of threshold stimuli. The individual cold points and warm points showed considerable differences in excitability, but these differences did not appear to be more than in normal areas. Thus the sensation of pain was called forth on very many spots, but the pain points did not lie so close together as in normal areas. There were painless spots of more than several square millimetres in area.

The disturbance of pain sense in general was so little marked that it might have been thought to be only anæsthesia to other sensations. For example, in some areas careful testing showed that pricks with a fine needle gave only pain without calling forth previous touch or pressure sensations. Thus, also, ice gave no sensation at all at first, then pain. A stimulus of 47° C. (116.6° F.) and upwards very quickly caused pain but no sensation of warmth; heat pain existed, therefore, without heat sensation. On some points this could be attained with a stimulus of 46° C. (114.8° F.) but hardly ever under 50° C. (122° F.). The stimulation according to the author was apparently due to pain organs; threshold pain with mechanical stimulus tested by von Frey's test hairs varied within the same limits as in normal areas. A cervical rib, the author maintains, has recently been shown to give rise, through pressure on the brachial plexus, to disturbances of sensibility and motility, which disappeared on resection. At the site of the disturbance the sensory paths must, in his opinion, all be arranged with regard to the peripheral distribution if one is to explain the fact that the two above-mentioned cutaneous nerves alone had been injured. As physiological experimentation shows that nerve fibres for flexion and extension of one portion of a limb are not evenly distributed within a nerve trunk but are arranged according to functions, the same arrangement may also hold for the sensory nerves with regard to the different paths which they are to follow within the spinal cord. In the present

case the majority of pain nerves were retained while others in great number had disappeared, for some areas of the skin even completely.

Cases of sensory election with such definite sensation losses as those recorded above are rare in affections of peripheral nerves, because these generally cause total and not partial sensory disorders. A sufficient number of cases exist, particularly of leprous neuritis, to show that the loss of pain is not uncommon in peripheral lesion. Biernacki, moreover, has called attention to the fact that pressure on the nervus ulnaris will cause a partial or total anæsthesia for pain and temperature with little diminution in sensitivity to pressure and with entire preservation of sensations of simple contact and with intact motor sensations and normal localization. Algesia, however, does remain in these cases as faradic stimulation will excite pain; this Laehr believes may be due to the conduction of the faradic current to pain nerves outside of the affected region which afterwards refer into the region of stimulation through the intact localization sense. It is also possible that compression may produce a functional depression of the pain nerves of such character that they are capable of being excited by a single form of stimulation and not by others, or else that the pain function is a physiological function of the other nerves. Pick, also, reports a case of traumatic lesion due to lying on the right shoulder, which presented a complete loss of temperature sensations in the forearm and hand, with pain and contact preserved. Cavazzani reports another exhibiting unequal loss of temperature and pressure, due to a wound, and which was cured on healing of the wound. J. B. Charcot reports a case presenting an acute swelling of the right hand with a dead feeling of the last two fingers followed by total analgesia and thermanæsthesia with some muscular atrophy. After surgical treatment, consisting of the removal of a cicatrix from the region of the palmar portion of the ulnar nerve, there was a complete return of sensation—first cold, then pain, then warmth, with a slow recovery from the muscular atrophy.

Laehr has collected a number of cases of lepra nervosa, exhibiting dissociated or elective sensory paralyses. Among these cases, Pitres reports one showing analgesic and thermanæsthetic spots existing conjointly over large areas, while Rosenbach, Sabrazes, Schlesinger, Jacoby, Marestant, Prus, and others report that analgesic spots may also exist alone or in combination with similar anæsthesiæ of greater extension. The last two also report conjoined tactile disturbances, and Thibierge-Bruhl reports a total analgesia and thermanæsthesia of the extremities, from the elbow and knee downwards.

These cases present the most direct proof of the functional loss of pain sensibility by one or more of the peripheral nerves. We may

therefore conclude that affections of the peripheral sensory neuron may cause the single loss of pain, which must be ascribed either to the cessation of the function of a specific pain nerve or to the loss of the pain function of one or all of the dermal nerves. While this creates a strong presumption in favor of the existence of a pain nerve, the proof of its existence cannot be regarded as conclusive without more direct anatomical demonstration, inasmuch as the action of pressure, as in Barker's case, and of neuritis, may possibly be to stop the pain-conducting function of all the peripheral nerves, leaving their other functions intact. This is a plausible hypothesis, at least, because the maximal nature of the pain stimulus permits the inference that the selective action of pressure on a nerve is to limit its range of response to the stimulus, without causing it to cease its functions entirely. For the practical purposes of diagnosis and study in cases presenting elective analgesia and hypalgesia, it seems to me justified by convenience and sufficiently in accord with the facts to speak of the functional disturbance as due to the disorder of a pain nerve, leaving it for future anatomical demonstration to ascertain whether the peripheral neuron, in this case the pain nerve, has but one or a number of sensory functions.

The Conduction Neuron or Tract of the Spinal Cord.

The evidence that I shall now present bearing on the existence of specific tracts or neurons in the spinal cord for the conduction of pain stimuli will be divided according to the methods of investigation into three sections. In the first, I shall speak of the experimental evidence for specialized pain tracts, in the second of the pathological evidence, and in the third of the evidence obtained from the phenomena of referred pains.

It is a well-known fact that even under normal conditions there is a delay in the arousal of pain when pain and other sensations are caused by one and the same stimulus. The cutting knife gives rise to a series of sensations of which touch is the first to arise in consciousness and pain probably last. Burkhart has called attention to this delay in the conduction of pain stimuli under normal conditions; Osthof long ago showed that in tabes dorsalis there was slowness of conduction for all sensory stimuli and that pain was more delayed than touch. Abnormal retardation in pain conduction, which can be referred to disorders of the spinal cord, seems to prove that the pain producing nerve stimulus differs from the other stimuli, either owing to its being conducted along different tracts or else to its meeting with conditions of increased resistance in the same tracts. Schiff was one of the first to maintain that the selective action of anæsthetics

upon pain required one to believe that there are different paths of conduction in the spinal cord for touch and pain, and the results of his experimentation went to prove that the former are in the white substance and the latter in the gray. Brown-Séquard, and Gad and Heymann, maintain that the gray matter in the cord alone is responsible for pain conduction. On the other hand, the importance of the lateral tract for pain has been emphasized by Ludwig, Woroschiloff, Gowers, and Sherrington, and these all unite in denying the decussation of the pain tract. Leyden and Goldscheider maintained that while anatomy and patho-anatomical facts show that localized pressure sensations are conducted through the posterior column of the same side, pain and temperature are conducted through the gray substance and its commissural fibres, perhaps also through the column cells, but that pain conduction is at all events double-sided. Bottarzi from experiments on the dog was led to believe that a lesion of one side of the cord produces lesion of motility and temperature, while a lesion of both sides produces hypalgesia. Turner's experiments upon apes seemed to show that all sensory tracts of the lower extremity crossed soon after entrance, whereas this is only the case for pain and temperature in the upper extremity; but Mott believes, also from experiments upon apes, that pressure and muscle sense cross while pain and heat are on both sides. Bechterew has shown on the dog that section of both lateral tracts and posterior halves of the cord produces analgesia only when the section is a little anterior to the pyramidal tract, and Holzinger corroborates him in this, maintaining further that the pain fibres are to be found in the middle third of the lateral column just anterior to the pyramidal tract.

Amid this conflicting evidence, the clear fact stands out of the recognition of a specialized conduction tract. The evidence seems to be in favor of locating this tract in the gray matter; pain and temperature seem to be associated locally in the cord, and the regions most favored by a majority of the authorities are the gray tracts and the lateral tracts. The evidence of decussation is quite conflicting. Laehr's conclusion, which will be considered later, harmonizes these conflicting opinions, in setting forth that the pain tract is first of the same side and subsequently crosses through the gray commissure to the opposite side, where it courses for a short distance in the gray column, finally entering the lateral column close to and somewhat anterior to the crossed pyramidal tract. Wundt is in accord with Schiff in accepting a specialized conduction tract. He believes, however, that this does not necessarily prove the existence of specialized pain nerves, but that the stimulus is conducted by all nerves to the spinal cord. Under normal conditions a stimulus, if of moderate intensity,

will pass up the white tracts without affecting the gray tracts, but if of greater intensity it flows over into the gray tract, giving rise to a stimulus that ultimately provokes a pain sensation. The gray substance therefore conducts peripheral stimuli of maximal intensity only, while the white substance transmits those of all intensities. This theory makes the pain sensation an associated sensation, due to the reference of part of the stimulus into the specialized tract in the gray substance.

Syringomyelia and other diseases of the spinal cord have made the phenomena of selective anæsthesia familiar. Ross, Starr, Sherrington, and Thorburn were among the first to call attention to the fact that anæsthesiæ of spinal origin were very different from those of peripheral origin, in that the latter were usually total or general over the area of distribution, whereas the former would show the dropping out of one or more qualities of sensation only. It will be unnecessary for me to present in any detail the familiar symptoms of syringomyelia. In a recent contribution on the disturbances in pain and temperature sensations, Laehr has taken occasion to make a *résumé* of cases of spinal lesions with loss of pain and temperature sensation, and has given them a critical examination in conjunction with an exposition of eleven new cases of his own, seven of which were cases of syringomyelia, two presented the Brown-Séquard complex, being one-sided lesions of the upper cervical cord and bulb, one was a double-sided affection of the dorsal cord, and the last a case of hæmatomyelia of the conus medullaris. The analysis of his own cases and a thoroughgoing examination of the entire literature seems to justify the conclusion that the extension of partial disturbances of sensation on the skin in syringomyelia and other disorders situated centrally in the spinal cord is characteristic of segmental disturbances; in this respect, these lesions present analogies with the cases of referred pains to be considered in the next following paragraphs. In the region of the trigeminus, also, the extension of the partial sensory disturbance corresponding to the central focus of the lesion seems to differentiate itself according to the territory of distribution of the nerve trunk and its three branches. The disturbance, in so far as it involves pain and temperature, is to be referred to the posterior gray substance at a definite level, although the segmental reference is not the same for partial as for total disturbance. Laehr further concludes that the tracts for pain and temperature enter the posterior horn on the same side, decussate later in their course to the other side, and subsequently unite into compact fibre bundles. proceeding centrad possibly in the lateral column. Their destruction in the gray substance generally causes a selective, most frequently

same-sided sensation disturbance; their destruction in the white substance gives rise to an anæsthesia for the entire lower opposite part of the body.

Despite conflicting evidence as to the relative frequency of sensation loss due to spinal-cord diseases, the cases are clearly enough indicative of the ease with which pain and temperature may go, while there is a relatively greater retention of pressure, touch, and motion sense.

The localization of the lesion is fraught with considerable difficulty, and there are many conflicting cases, but the analysis of Laehr seems to harmonize most of these. Charcot and Gombault report a case of luetic disease of the cord with analgesia of the entire left side showing an injury of the gray matter of the pain fibres after crossing. Kiär reports an analgesia due to lesion of the cross-fibres in the gray matter. Gowers has recorded a case in which a unilateral hemorrhage into the lateral column and gray substance of the upper cervical cord produced analgesia and thermanæsthesia. In this case there was a complete loss of pain on the opposite side without disturbance of touch. Similar cases are reported by Vucetis, Beevor, Williamson, Eulenburg, and Determan. Schlesinger compares the sensibility disturbances in syringomyelia with the Brown-Séquard symptom complex, and arrives at a localization of pain and temperature tracts in the cord which Laehr's hypothesis fairly well covers.

Referred pains are familiar to many. Most of us have either suffered ourselves or had friends who have suffered from the "ice-cream nerve" at least, that severe pain between the eyes after eating of cold substances. This is a referred pain, having its seat of origin in the stomach, but given a false reference perhaps through the local association of the afferent nerves from the stomach with sensory nerves in the upper cord distributing to the face. Munro reports a case of pain in the front of the chest that could be induced by friction in the forearm. Mitchell reports another in which rubbing or pinching a mole on the leg gave rise to a sharp pain in the chin. Stroking the back of the neck in some persons causes a pain and shivering down the spine, and I have known apparently normal subjects who obtained definitely localized pains through scratching or pinching remotely situated areas. A case in point that has just come to my notice presents a man, twenty-eight years of age, who has had no severe illness, no headaches, no neuralgias or rheumatism, no tendency to hallucination or to hysterical manifestations, who can, by pinching the back of the neck on the right side, and the lateral portion of the neck up to the right ear, produce a pain that is localized in the dorsal regions of the back on the left side, although he

says that sometimes it appears on the right side also. The pain is often also referred into the arms. Scratching the area on the neck also produces gooseflesh above and to the right of the right eye. Irritation of the collar, particularly if he has a pimple on the neck, will give rise to this referred pain. Sometimes the sensation is not noticeably painful, it is merely one of itching. This pain cannot always be provoked in this way, and he thinks that it cannot be so easily aroused at the present time as it was five years ago when it first came to his notice. A pimple elsewhere on the body will also at times produce pains referred to other regions, but they are never referred into the legs, nor will a stimulus applied to the legs give rise to referred pains anywhere in the body.

A large number of cases are on record of referred pains due to pathological conditions. Mitchell reports one of a shell wound of the right leg which at once gave rise to a burning pain in both feet and in the right arm and right pectoral regions. Another shell wound of the outer side of the left thigh produced an immediate reference of pain to the same area on both legs so that the man thought he was shot through both thighs. This is a cross symmetric reference somewhat similar to the cases that have been reported under the name of allocheiria. Case IV. of Hutchinson's series is cited by Mitchell as being an injury to the median and ulnar nerves giving rise to pain in the opposite hand. Pirogoff has a report of the same sort of cross reference from a wound. Another case of Mitchell's somewhat approaches those of reference in visceral disease, in that a testicle wound aroused a pain which was alone referred to the back. Hilton was the first to call attention to these pains; latterly they have been carefully studied by a number of investigators.

A most instructive form of reference is that condition to which Obersteiner has given the name allocheiria. The phenomenon presents a pain or other sensation which is referred to a symmetric part of the body. This was first observed by Obersteiner affecting the hands, in consequence of which he applied to it the name of allocheiria; but it has also been found in the legs. Obersteiner's original contribution reported four cases of his own and five collected from other sources, one being hysterical and the others occurring in the course of tabes dorsalis. Gowers mentions a patient, the subject of tabes, who referred a touch to corresponding points on both sides of the body at the same time. Gay reports a recent case of partial sensory paralysis due to diphtheria in which slight touches were not felt at all, but firmer ones were referred to exactly symmetric parts of the opposite side. There was no hesitation

in localizing; painful sensations were not tested, but the kinæsthetic sensations of all four extremities were entirely gone, so that the limbs could be put into all sorts of grotesque attitudes without the patient being aware of anything but the warmth of the hands. All cases of allocheiria, according to Gay, are accompanied by considerable ataxia and profound disturbance of common sensations. These symptoms, in his opinion, are probably due to an affection of the posteromedian columns with the posterior root zone, and include the sensory decussating fibres. But the latter appear to be still intact as in no case was the sense reported entirely lost. Gay believes that we should explain such cases by supposing that the common sensory stimuli find their usual channel closed against them, and if of greater than normal intensity, are conducted upwards by the non-decussating fibres, referring in consequence to the opposite side of the body. Dumontpelier is reported to have found that by etherizing one arm in a particular position he was able to open painlessly an abscess situated in the corresponding area of the opposite side. Psychological experimentation also has shown that when practice in a given part of the body causes an increase in the sensory discrimination of that region, the symmetric region on the other side of the body also shows some improvement. Cattell showed that a stimulus applied to a symmetric portion of the body with reference to the reacting member gives a shorter reaction time than when it is applied elsewhere. These cases alone prove that pain behaves as do other sensations when crossed reference is observed. That symmetric parts of the body are thus shown to be more closely connected with one another than with other portions of the body, and that the lesion generally affects coördination and muscle sense to a considerable extent, suggest that the basis for this reference of pain, and perhaps for all reference, is an associated involvement of the posterior columns, which mediate sensations of touch and movement.

More than ten years ago, Dana made a careful study of referred pain of the cephalic extremity, and recently Head has published a series of articles whose scientific value cannot as yet be estimated, but whose principal conclusions have such important bearing on the nature of pain production and pain reference that they deserve special consideration. The early parts of Head's contribution were concerned with the disturbances of sensation, particularly of pain in visceral disease, and were based upon the belief that some coördination could be found between the distribution of such referred pains and that of herpes zoster. Visceral disease is marked either by a pain or by a tendency to pain, that is, hyperalgesia or tenderness, which may be either local—that is, situated near the organ of excitation—or referred

to regions only remotely connected with such organ. Head believes that he is able to mark out fifteen well-defined areas, and that a study of the cases of herpes zoster shows that the manifestations of these conditions appear upon exactly the same areas as those which are tender in visceral disease. So far as the visceral diseases alone are concerned, these areas might correspond to any level of the spinal cord or indeed of the entire central nervous system, or they might represent the supply of the posterior roots or peripheral nerves. It is Head's opinion that these areas represent the nerve supply for heat, cold, pain, and trophic distribution of a given spinal segment at some particular level only. The viscera receive their sensory fibres from the same segment of the spinal cord from which the fibres of the somatic sensory areas arise, to which the pain is referred. Certain organs have a tendency to refer into the supply of certain nerve plexuses; thus the lungs, heart, liver, intestines, and stomach tend to refer into that of the cervical plexus and cranial nerves. There are two groups of segments in the spinal cord which, however, are in no connection with the sensory fibres from the viscera. These are the fifth, sixth, seventh, and eighth cervical segments forming one group, and the second, third, and fourth lumbar constituting the other.

Several forms of the progressive spreading of pain through continuation of visceral irritation are manifested. After pain has remained localized in any given region the condition of the central nervous system becomes profoundly altered and a tendency manifests itself for both pain and tenderness to spread, in some cases even to the opposite side of the same spinal level. A somewhat different form of generalization or spreading is the diffusion or spreading that is seen in toothache. A tooth alone may be first affected, but the pain becomes gradually accentuated by neuralgia as the toothache continues, that is, the pain is referred to the face. This is accompanied by distinct cutaneous tenderness on the face corresponding to the tooth affected. If the patient be anæmic or the pain remains untreated bodily health becomes affected, the pain and tenderness spread until the whole of one-half of the head and even the neck has profound tenderness over areas which bear no relation to the affected organs. The spreading from one organ to an organ associated in function, or rather to that of an area corresponding to such organ, gives a third form of generalization; thus the uterus and mammæ are functionally closely related, and pain and tenderness may spread from the area of the former to that of the latter. The order in which generalization takes place also indicates a condition of the cord that may be spoken of as the relative specific resistance of the centres for the sensory impulses from various organs. There is a tendency, for example, to refer pain into an

organ of diminished resistance; thus during menstruation, a hypermetrope will suffer from agonizing headache which may spread to the neck. Any cause, even a mental one, tending to reduce the resistance of the central nervous system will thus promote generalization.

It is Head's opinion that the localization of sensation is not a psychical but a physical association. The final result of any given stimulus, say it be one acting upon the foot, appears in consciousness as the centre of a conscious field which extends over a more or less wide area; that is to say, it is not only the point to which the stimulus is applied which comes into consciousness but the whole foot or the whole leg. If the power of perceiving pain is diminished on the foot it is referred higher up or possibly to the corresponding point on the opposite foot. The same rule applies to each form of sensation. If the sensation of touch is affected, pain and heat and cold being normal, touch only will be referred, while heat, cold, and pain are rightly localized. Head obtained records in which only one form of the four sensations was wrongly localized, while the others were localized over the actual point of stimulation. In all such cases of wrong localization the particular sensation which is wrongly localized must be diminished and must not be absolutely lost over the affected area. If it is lost the sensation is simply not perceived. To continue nearly in Head's words: "Whenever an area representing one or more spinal segments on one side of the body is partially anæsthetic while the sensation of the areas representing the same segment on the other side is normal, a stimulus to the skin over the affected areas is referred to the corresponding point on the normal side. If, however, the segments on both sides are affected the stimulus is referred to the area corresponding to the area next above or next below the affected segments in their origin from the cord. The internal organs are structures of low sensibility to touch location owing to their inaccessibility. It is therefore not to be wondered at if the maximum of pain is not felt in the organ affected. A painful stimulus from an internal organ is conducted to that segment of the cord at which its sensory nerve enters. Here it comes into close connection with the fibres for painful sensation from the surface of the body, also arising from the same segment. As the sensory and localizing power of the surface of the body is enormously in excess of that of the surface of the viscera, an error of judgment occurs, the diffusion area being accepted by consciousness, and the pain referred to the surface of the body instead of to the organ actually affected. Thus both allocheiria and visceral reference depend for their appearance on the law that where a painful stimulus is applied to a part of low sensibility in close central connection with the part of much greater sensi-

bility, the pain produced is felt in the part of higher sensibility rather than in that of lower sensibility to which the stimulus is actually applied."

Visceral pain always produces exaggeration of the sense of pain and sometimes exaggeration of the sense of heat and cold in the skin, but never, in Head's experience, hyperæsthesia proper. As in syringomyelia, pain, heat, and cold are profoundly disturbed, with the development of cutaneous trophic lesions, while the tactile sense is unaffected.

Thus the central connections of the pain fibres from the skin and viscera are closely connected with one another, while the central connections of the nerves for heat and cold, and for trophic disturbance in the skin must also be in somewhat close asssociation, though probably not actually connected. On the other hand, the nerves for touch from the skin are widely separated centrally from those of pain. As to their intraspinal paths Head does not speak.

Mackenzie substantiates Head's results in the main, though differing from him in many important details. The former contributes other evidence bearing on these segmental relationships, such as the reflex alleviation of pain in the central organs by counter-irritation of the periphery. His study of the pilomotor, or goose-skin reflex, raises some doubt as to the definiteness of the separation of the different peripheral areas as outlined by Head. The pilomotor nerves over a limited area, like the nerves of special sense, may be rendered abnormally sensitive in diseases of the internal organs. A study of the spread of such excitation from any given centre in a cord seems to indicate that the normal path is longitudinal rather than transverse in direction, following the disposition of the peripheral nerve in the longitudinal or vertical direction on the dorsal, lateral, and ventral surfaces of the body. It is of some interest to the psychologist at least that the sensation of gooseflesh may be produced without any of its characteristic appearances, and may indeed be produced in regions where there are no pilomotor nerve endings. In an excellent case the subject was able to mark out the peripheral distribution of the nerve from the feeling that he had when the skin over the ribs was touched. Thorburn also supports Head's main contention, and White, in a study of the exact sensory effects produced by lesion of the spinal cord, reports a case of cutaneous analgesia without anæsthesia due to compression of the cord at the point of origin of the eighth dorsal nerve in which the areas affected corresponded closely to those of Head.

The examination of the literature that has here been made seems to justify certain conclusions in regard to pain conduction in the

spinal cord. The evidence seems to point to certain tracts in the cord possessing specific pain function. The gray matter of the cord is a specialized tract for the conduction of pain stimuli for a short distance above the segment of the cord which contains the nerves of the peripheral distribution. This tract, whatever its exact course may be, can be considered a spinal pain organ. The tracts possibly decussate, cross to the opposite gray matter, and finally pass on into the lateral column just anterior to the crossed pyramidal tract. The peripheral nerves stimulate this central pain tract or organ under certain conditions that depend upon the intensity, the duration, mode of application, and intermittency of all peripheral stimuli. In common with the nerves from the periphery, the viscera also refer excessive stimulation into this pain organ of the spinal cord. Locality is an ascription that is given to the pain or other sensation, not something residing in the sensation *per se*. The "local color" or "local sign" of pain is dependent upon the presence of other sensations, most likely those of movement which are so closely associated with specific touch. These two pass up together in the posterior column, while pain and temperature together course for a while at least in the gray column; thus there is less likelihood to be disturbances of touch or common sensation than of pain and temperature. The giving of locality to a pain depends upon the associated activity of the tracts for touch or movement, both of which at each spinal segment seem to be conjoined with the pain and temperature tract. The reference of a given pain into a peripheral part of the body, whether that pain be due to a stimulus from without the body or from within, is evidence not only of the excitation of pain tracts from that region but of the excitation of some other nerves than pain that give locality to pain stimuli coming from the region of their application. In peripheral reference, therefore, I believe that the pain tract must be assumed to be excited in visceral disease, part of the stimulus referring into the pain tract, occasionally also into the temperature tract, but the other tracts may also be acted upon by the stimulus, and these contribute to the localization of the pain quality. These tracts must be supposed to be conjoined at each segment where they enter the cord, but afterwards to separate. In each segment, therefore, we have a pain tract capable of receiving stimuli from the afferent fibres of the area of segmental distribution as well as from the internal organs, trophic nerves, and so on. The facts displayed above show that it is relatively easy for nerves to refer into: (1) A symmetric area; (2) functionally associated organs; (3) the segmental area; (4) longitudinally related areas. Subsequent investigation must reveal the anatomical association of the neurons of the spinal cord,

which subserves the varied phenomena of pain reference, and also furnishes the basis for a distinction between the parts played by cortical and by spinal association in the localization of pain and other forms of sensation.

The Path for the Conduction of Pain Stimuli in the Brain.

The character of the phenomena produced by irritative lesions of the brain stem is of interest to the diagnostician, no less than to the psychophysiologist. In the crura cerebri we are above the regions of segmental association and reference. If pain can be evoked by stimulation of the "carrefour sensitif," the evidence would seem to be clear for the existence of a cortical pain centre, and the areas of reference of such pain ought to supply valuable data for the study of the location of this area and of associated areas in one or both hemispheres. The examination of cases cannot be said to give any clew as to the location of a sensory centre of pain. They are too few in number, and they demand a more careful examination than has yet been accorded them. They demonstrate, however, that central pains may be produced by irritation or compression of the conduction tract of the thalamus, and that such pains may be peripherally referred.

Such pain may exist alone as a spontaneous pain or, as some would perhaps call it, an hallucination of pain. It may coexist with hyperæsthesia or hypæsthesia, or hyperalgesia only may exist.

Möbius denies the possibility of producing pain through an affection of the central conduction tract. Despite this opinion, however, a number of cases have been brought forward by Edinger, Biernacki, and others, showing the possibility of the arousal of pain through the agency of irritative lesions of the thalamus. Edinger's case was the first clearly to demonstrate the possibility of such central pain production. The first symptom manifested in this case was a boring, tearing pain, accompanied by motor weakness occurring the first day after the attack. The motor weakness improved, but the pain remained, leading to suicide two years after the initial lesion and after a number of attempts. The lesion in this case lay close to the posterior third of the posterior segment of the internal capsule. The part of the inner capsule which must contain the central feeling tract remained intact, viz.: the middle and posterior third of the thalamus. The feeling tract, therefore, lay close to the field of degeneration, but was not itself directly injured. The direct contact of the sensory tract with the diseased tissue furnishes the anatomical possibility for the production of the hyperæsthesia and the pain in the cross parts of the body. Biernacki reports a case that is the alter ego of Edinger's. A woman of fifty-four presented a symptom complex of headache,

vertigo, coughing, with severe pain for a number of years in the right side of the body, and for five years weakness of the right side coupled with involuntary movements of the right hand and sometimes of the right leg. On movement of the parts, pain could be produced throughout the arm and leg, but when at rest the pain was chiefly in the elbow and knee. The touch sense was intact, although there was slight hypæsthesia. The localization ability and the temperature and muscle senses were intact excepting a slight numbness to cold. There was a high degree of hyperalgesia to pressure, percussion, and all passive movements. A vertical section through the left pulvinar of the thalamus showed, on the border of the middle and posterior third, a round pea-sized cyst filled with fluid interwoven with connective tissue, and in the immediate neighborhood of the inner capsule, which contains the sensory tracts that constitute the so-called "carrefour sensitif," and which, according to Türck, Charcot and his school, is devoted in its most posterior part to touch and the muscle sense. To this situation of the lesion is to be ascribed the induction of the pain by irritation of the cerebral conduction tracts, inasmuch as hysteria and other possible explanations were excluded.

Greif reports a thalamus lesion in a woman of seventy-four with a symptom complex consisting of choreiform movements in the left side, especially in the left arm, together with hyperæsthesia and tearing pains in the same arm and to a less degree in the left leg. The chorea disappeared in fourteen days, but the pain remained until death. The pains in this case were referred to the opposite side of the body, as a lesion was found in the left thalamus in the neighborhood of the corpus subthalamicum just over the crura cerebri, which was looked upon as the cause of the excitation symptoms. Lauenstein reports a case of injury in the right thalamus with hemichorea of cerebral origin and severe pain in the moved limb. Apparently there was no autopsy. Gowers and Nothnagel make mention of hyperæsthesia and spontaneous peripheral pain associated together. Mann reports a case of softening of the oblongata in which for six years after the onset of symptoms the patient had headache and burning pains in the left half of the body and right half of the face; there was paresis in the region of the bulbar nerves with general hypæsthesia in the hyperalgesic region, but in this case the central pains were the most emphatic symptom; these remained for five and one-half years. Cold was called cool and hot was called warm, but passive and active touch were intact. The lesion was close to the conduction tract, but there was no complete break in the tract, although there was a separation of some fibres. Leyden's case of acute bulbar paralysis presented painful formication of the upper extremity, while Marot's case

of a solitary tubercle in the right half of the pons presented strong neuralgic pains in the left extremities with complete anæsthesia of the skin, the so-called næsthesia dolorosa. Duchek's tumor of the pons produced pulling pains, formication, going to sleep of parts, but in this case the pain soon ceased and hyperæsthesia of the face followed. Nothnagel cites observations of Guinon, Martinot, and Eichhorst of tumors and softening in the pons, causing from the first day a passing hyperæsthesia of the opposite halves of the body.

The symptoms here produced by central irritation are somewhat analogous to those which Herzen and Goldscheider have observed in compression of the nerve trunk, and which in one or two cases have been found in compression of the spinal cord. The pain may exist as spontaneous pain alone; it may exist with hypæsthesia and even hypalgesia of the part, or with hyperæsthesia, or the pain may exist as a hyperalgesia which requires some stimulus to evoke it. Paræsthesiæ also accompany affections of the sensory tract; in such cases cold and heat are first affected, touch and muscle sense but rarely, while formication, jerking, and going to sleep are often mentioned as present.

Cortical Pain.

Both Edinger and Biernacki suggest that, in some central affections due to irritation, spontaneous pain may exist as the only symptom, and that such pain may sometimes be of cortical origin. They regard the painful aura in epilepsy as furnishing some evidence of cortical pains, as do also the paræsthesiæ of cold and other abnormal sensations that must be ascribed to cortical regions. Edinger believes that future investigation must show how far the severe pains of hypochondriacs and the pains of hysterics are of central origin. Claiborne presents a case of subjective pain, while Starr reports one of "subconscious pain," which he successfully treated by hypnotism. This case was in a girl of nineteen, whose only symptoms were bilateral pains of the arms, which, though not at all sharp or shooting, were severe enough to make her uneasy and incapable of steady occupation. They were worse when she woke up in the morning, and frequently were severe enough to awake her from sleep. Except some anæmia, she had no other symptoms. The pain is described by Starr as "hysterical, subjective, mental, delusional, and not to be ascribed, therefore, as due to any physical external cause." From a psychophysiological standpoint, serious objection may be made to this description of the pain and to the conclusion that it is not to be ascribed as due to any physical cause. The pain certainly existed as

an actuality in this girl's mind, and it must have had some cause, irritative or otherwise, in some part of the central nervous system. It is in harmony with the views of the best authorities to consider this as a pain of cortical origin. Richet suggests that absence of pain also is a symptom of central lesion, and maintains that there is a good deal of evidence in favor of a localized sense organ in the brain. Mitchell reports that some women remain for years without the peripheral pain sense, although the general health is unimpaired, while the internal organs still feel pain and all forms of skin sense are as keen as ever. A somewhat similar case is that of a "professional painless man," who was for some time under my observation, and who showed himself capable of making himself insensible to pain whenever he wished to do so. He had worked for some time in a dime museum as the human pin cushion, and could be cut with a knife or stuck with pins or needles without showing the slightest sign of pain. I have known him to hold a red-hot half dollar in his hand without wincing until it had burnt itself deep into the flesh. It is impossible to say positively whether this subject inhibited the expressive movements of pain, that is, the external signs of pain, or whether he inhibited the pain itself. If his own statements can be trusted, he felt pain on ordinary occasions when he had not made up his mind to be insensible to pain, but he reports that, when he had once decided not to feel the pain of the stimulus, the pain was no longer felt. It was not stoically endured. Moreover, there were areas of the skin which he could not render insensible to pain in this way. I am therefore inclined to believe that he inhibited the sensation of pain and not its external manifestations.

This case and many cases of hysterical pain present phenomena analogous to those of the behavior of subjects in a hypnotic state as respects pain. It has been my experience that the pain sense is most easily affected in light hypnosis, whether in the direction of hypalgesia or in that of hyperalgesia. Certainly with average normal subjects, motor phenomena and pain disorders are much more readily produced by hypnotic suggestion than are disorders of the other senses. The behavior of the pain sense in the hypnotic state and in hysteria is one of considerable importance for the localization of a cortical pain centre, inasmuch as these phenomena must in great part be ascribed to disturbances of the cortex of the cerebral hemisphere.

Despite this evidence of the existence or absence of pain due to cortical conditions, no anatomical or physiological evidence of a sufficiently satisfactory character is forthcoming to assign a locality to the pain centre in the cerebral hemisphere. As has already been

tract in the spinal cord as the spinal nerve organ of pain, which, together with the hypothetical specialized cortical centre, constitutes the specific organ of pain.

9. Any part of this central pain organ may be stimulated in the cortex or below it, either by stimuli discharging into it through normal physiological processes, by spinal or cortical association, by irritation due to disease, and perhaps by a vascular disturbance within the central nervous system.

Thus pain may be a sensation of purely central nervous origin. The arousal of pain by stimuli and its presentation in consciousness along with other sensations may be explained by the simultaneous association of pain with other forms of stimulation—an association that may take place at any level of the nervous system. When such association takes place in the cortex we have conscious association, the connection of pain with other sensations, with percepts, or with ideas. Many persons associate colors with sounds. This phenomenon, known as colored audition or pseudochromæsthesia, and which has received careful study since its first investigation by Galton, is explained as a cortical association. In a like manner the association of pain with other sensations may be explained as due to cortical or spinal association.

In one important respect the association of pain with other sensations (viz., those due to intense stimuli) differs markedly from the association of colors with sounds. The former is normal and constant in the human organism, while the latter, although it has been found to occur with surprising frequence, is yet of abnormal and inconstant character. It is only in cases of referred pains due to irritation of the central organ itself that abnormal pain associations are found, presenting a complete analogy with the phenomena of colored audition. The universality of pain association with sensations of maximal intensity is explainable anatomically and physiologically by the discharge of intense stimuli carried by all peripheral nerves into the central pain organ; that this central pain organ has no peripheral nerves of its own, possessing a specific pain function and no other, can receive satisfactory explanation only from biological considerations of the significance of pain as a warning against dangerous stimuli of the environment. This fact would suggest an early phylogenetic development of a pain sense in the organism; in fact, the pain sense may have been the first of all the special senses. The sensory neuron, in the course of phylogeny, seems to grow inwards to the central nervous system, and it may not be advancing too far along the road of hypothetical speculation to suppose that, while at one time the pain organ may have been part centrally and

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